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NEWS 4	JUN 26	NUTRACEUT and PHARMAML no longer updated
NEWS 5	JUN 29	IMSCOPROFILE now reloaded monthly
NEWS 6	JUN 29	EPFULL adds Simultaneous Left and Right Truncation (SLART) to AB, MCLM, and TI fields
NEWS 7	JUL 09	PATDPAFULL adds Simultaneous Left and Right Truncation (SLART) to AB, CLM, MCLM, and TI fields
NEWS 8	JUL 14	USGENE enhances coverage of patent sequence location (PSL) data
NEWS 9	JUL 27	CA/CAplus enhanced with new citing references
NEWS 10	JUL 16	GBFULL adds patent backfile data to 1855
NEWS 11	JUL 21	USGENE adds bibliographic and sequence information
NEWS 12	JUL 28	EPFULL adds first-page images and applicant-cited references
NEWS 13	JUL 28	INPADOCDB and INPAFAMDB add Russian legal status data
NEWS 14	AUG 10	Time limit for inactive STN sessions doubles to 40 minutes
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NEWS 16	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS 17	AUG 24	CA/CAplus enhanced with legal status information for U.S. patents
NEWS 18	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
NEWS 19	SEP 11	WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus

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                                                           ENTRY    SESSION
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=> s ?setron and (?mucosal)
L1          396 ?SETRON AND (?MUCOSAL)

=> s l1 and py<=2003
L2          270 L1 AND PY<=2003

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3          136 DUP REM L2 (134 DUPLICATES REMOVED)

=> s l3 and ondansetron
L4           52 L3 AND ONDANSETRON

=> d l4 ibib abs 1-52

L4   ANSWER 1 OF 52  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:         2005:316336  CAPLUS
DOCUMENT NUMBER:          142:360872
TITLE:                    Buccal aerosol sprays or soft gelatin capsules for
                          biologically active agents such as diazepam
INVENTOR(S):              Dugger, Harry A., III; Abdel-Shafy, Mohammed
PATENT ASSIGNEE(S):       Novadel Pharma Inc., USA
SOURCE:                   PCT Int. Appl., 57 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   19
PATENT INFORMATION:

     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
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     WO 2005032517        A1    20050414    WO 2004-US31798        20040927
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH,
             CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD,
             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM,
             AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK,
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EP 1036561	A1	20000920	EP 2000-109357	19971001	<--
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EP 1952802	A3	20090617			
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EP 2042161	A1	20090401	EP 2008-20267	19971001	
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US 20050163719	A1	20050728	US 2003-671709	20030929	
CA 2582007	A1	20050414	CA 2004-2582007	20040927	
EP 1675566	A1	20060705	EP 2004-789150	20040927	
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JP 2009079060	A	20090416	JP 2008-266598	20081015	
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PRIORITY APPLN. INFO.:	US 2003-671709	A	20030929
	EP 1997-911621	A3	19971001
	JP 2000-513555	A3	19971001
	WO 1997-US17899	A2	19971001
	US 2000-537118	A2	20000329
	US 2002-230060	A2	20020829
	WO 2004-US31798	W	20040927

AB Buccal aerosol sprays or soft gelatin capsules are developed using polar and non-polar solvent, providing rapid absorption of biol. active compds., such as diazepam, through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise (i) aqueous polar solvent, diazepam, and optional flavoring agent; (ii) aqueous polar solvent, diazepam, optionally flavoring agent, and propellant; (iii) non-polar solvent, diazepam, and optional flavoring agent; (iv) non-polar solvent, diazepam, optional flavoring agent, and propellant; (v) a mixture of a polar and a non-polar solvent, diazepam, and optional flavoring agent; and (vi) a mixture of a polar and a non-polar solvent, diazepam, optional flavoring agent, and propellant. For example, a propellant-free diazepam formulation in a polar solvent contained diazepam 2%, propylene glycol 50, EDTA 0.02, benzalkonium chloride 0.02, taste mask 0.1%, glycerol 0.5%, Tween 80 0.5%, water 2%, and ethanol to 100%.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 2 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2004:569660 CAPLUS
DOCUMENT NUMBER:	141:94376
TITLE:	Buccal, polar and non-polar spray containing atropine
INVENTOR(S):	Dugger, Harry A., III; Abd El-Shafy, Mohammed
PATENT ASSIGNEE(S):	USA
SOURCE:	U.S. Pat. Appl. Publ., 21 pp., Cont.-in-part of U.S. Ser. No. 230,085. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	19
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 20040136915	A1	20040715	US 2003-671719	20030929	
WO 9916417	A1	19990408	WO 1997-US17899	19971001	<--

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RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

EP 1036561	A1	20000920	EP 2000-109357	19971001 <--
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EP 1952802	A2	20080806	EP 2007-23005	19971001
EP 1952802	A3	20090617		
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
EP 2042161	A1	20090401	EP 2008-20267	19971001
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
US 20030095926	A1	20030522	US 2002-230085	20020829 <--
US 20070048229	A1	20070301	US 2006-443260	20060531
JP 2009079060	A	20090416	JP 2008-266598	20081015
JP 2009149675	A	20090709	JP 2009-41207	20090224

PRIORITY APPLN. INFO.:	WO 1997-US17899	A2 19971001
	US 2000-537118	A2 20000329
	US 2002-230085	A2 20020829
	EP 1997-911621	A3 19971001
	JP 2000-513555	A3 19971001
	US 2003-671719	A3 20030929

AB Buccal aerosol sprays or capsules using polar and non-polar solvent have now been developed which provide atropine for rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise formulation I: aqueous polar solvent, atropine, and optional taste mask and/or flavoring agent; formulation II: aqueous polar solvent, atropine, optionally flavoring agent, and propellant; formulation III: non-polar solvent, atropine, and optional flavoring agent; and formulation IV: non-polar solvent, atropine, optional flavoring agent, and propellant; formulation V: a mixture of a polar and a non-polar solvent, atropine, and optional flavoring agent; formulation VI: a mixture of a polar and a non-polar solvent, atropine, optional flavoring agent, and propellant.

L4 ANSWER 3 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2004:182257 CAPLUS
DOCUMENT NUMBER:	140:223296
TITLE:	Intravaginal or transmucosal delivery of antimigraine and antinausea drugs
INVENTOR(S):	Pauletti, Giovanni M.; Soderstrom, Richard; Ritschel, Wolfgang A.
PATENT ASSIGNEE(S):	USA
SOURCE:	U.S. Pat. Appl. Publ., 24 pp. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	12

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20040043071	A1	20040304	US 2003-600849	20030620
AU 765269	B2	20030911	AU 2001-54192	20010703 <--
US 20050249774	A1	20051110	US 2005-126863	20050510
US 20050276836	A1	20051215	US 2005-180076	20050712

PRIORITY APPLN. INFO.:

US 2002-390748P	P	20020621
US 1997-49325P	P	19970611
US 1998-79897	A2	19980515
AU 1998-76976	A3	19980610
US 1999-249963	A2	19990212
US 1999-146218P	P	19990728
US 2000-626025	A2	20000727
US 2002-226667	A2	20020821
US 2003-349029	A2	20030122
US 2003-600849	A2	20030620
US 2004-587454P	P	20040712
US 2005-126863	A2	20050510

AB A method, composition and device for intravaginal mucosal or transmucosal delivery of antimigraine and/or antinausea drugs to a female subject for treatment of migraine and other diseases accompanied by or associated with nausea and vomiting. A mucoadhesive composition comprising antimigraine or antinausea drugs, mucoadhesive agent, penetration enhancer or sorption promoter and a hydrophilic or lipophilic carries. An intravaginal device for delivery of antimigraine or antinausea drugs. Vaginal suppositories comprising a dose of 50 mg/suppository were prepared The composition of the pharmaceutical excipients in these formulations was Suppocire AS2X 66, HPMC 1.5, Transcutol 15, and water 15%.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L4 ANSWER 4 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:892541 CAPLUS
DOCUMENT NUMBER: 139:369733
TITLE: Multi-phasic delivery via transmucosal absorption of antiemetic medicaments
INVENTOR(S): Pinney, John M.; Cone, Edward J.
PATENT ASSIGNEE(S): NPD LLC, USA
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003092591	A2	20031113	WO 2003-US13255	20030430 <--
WO 2003092591	A3	20040325		

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RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2003241322	A1	20031117	AU 2003-241322	20030430 <--

PRIORITY APPLN. INFO.:

US 2002-376263P	P	20020430
WO 2003-US13255	W	20030430

AB The present invention concerns a composition for oral administration of an active for suppressing nausea and vomiting. The composition comprises a carrier, an antiemetic active, and a buffer. The carrier may be a gum, a lozenge, a candy or a tablet suitable for administration in an oral cavity. The buffer is water-soluble, and facilitates bi-phasic release of the active for transmucosal absorption. The method of

delivering the antiemetic active in a bi-phasic manner is also provided.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 5 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2003:396255 CAPLUS

DOCUMENT NUMBER: 138:406917

TITLE: Buccal sprays or capsules containing drugs for treating disorders of the gastrointestinal or urinary tracts

INVENTOR(S): Dugger, Harry A., III

PATENT ASSIGNEE(S): USA

SOURCE: U.S. Pat. Appl. Publ., 14 pp., Cont.-in-part of U.S. Ser. No. 537,118.
CODEN: USXXCO

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 19

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030095926	A1	20030522	US 2002-230085	20020829 <--
WO 9916417	A1	19990408	WO 1997-US17899	19971001 <--
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EP 1036561	A1	20000920	EP 2000-109357	19971001 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
EP 1952802	A2	20080806	EP 2007-23005	19971001
EP 1952802	A3	20090617		
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
EP 2042161	A1	20090401	EP 2008-20267	19971001
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CA 2497112	A1	20040311	CA 2003-2497112	20030827
WO 2004019910	A2	20040311	WO 2003-US26854	20030827
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JP 2006506342	T	20060223	JP 2004-531570	20030827

NZ 539285	A	20071026	NZ 2003-539285		20030827
NZ 561128	A	20071130	NZ 2003-561128		20030827
US 20040136914	A1	20040715	US 2003-671717		20030929
US 20040136915	A1	20040715	US 2003-671719		20030929
US 20050025716	A1	20050203	US 2004-928996		20040827
US 20060198790	A1	20060907	US 2006-429953		20060509
US 20070048229	A1	20070301	US 2006-443260		20060531
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PRIORITY APPLN. INFO.:			WO 1997-US17899	A2	19971001
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			JP 2000-513555	A3	19971001
			US 2002-230085	A	20020829
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			US 2003-671717	A3	20030929
			US 2003-671719	A3	20030929
			US 2006-429953	B1	20060509

AB Buccal aerosol sprays or capsules using polar and non-polar solvent have now been developed which provide biol. active compds. for rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise formulation I: aqueous polar solvent, active compound, and optional flavoring agent; formulation II: aqueous polar solvent, active compound, optionally flavoring agent, and propellant; formulation III: non-polar solvent, active compound, and optional flavoring agent; and formulation IV: non-polar solvent, active compound, optional flavoring agent, and propellant. A lingual spray contained famotidine 7-20, water 5-10, L-aspartic acid 5-10, polyethylene glycol 50-85, and flavors 2-5%.

L4 ANSWER 6 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2003:334375 CAPLUS
DOCUMENT NUMBER:	138:343878
TITLE:	Buccal sprays or capsules containing drugs for treating an infectious disease or cancer
INVENTOR(S):	Dugger, Harry A., III
PATENT ASSIGNEE(S):	USA
SOURCE:	U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S. Ser. No. 537,118. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	19
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030082107	A1	20030501	US 2002-230080	20020829 <--
WO 9916417	A1	19990408	WO 1997-US17899	19971001 <--
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JP 2006502150	T	20060119	JP 2004-531575		20030827
US 20050142069	A1	20050630	US 2004-929001		20040827
JP 2009079060	A	20090416	JP 2008-266598		20081015
US 20090186035	A1	20090723	US 2009-351179		20090109
JP 2009149675	A	20090709	JP 2009-41207		20090224
PRIORITY APPLN. INFO.:			WO 1997-US17899	A2	19971001
			US 2000-537118	A2	20000329
			EP 1997-911621	A3	19971001
			JP 2000-513555	A3	19971001
			US 2002-230080	A	20020829
			WO 2003-US26860	W	20030827

AB Buccal aerosol sprays or capsules using polar and non-polar solvent have now been developed which provide biol. active compds. for rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise formulation A: aqueous polar solvent, active compound, and optional flavoring agent; formulation B: aqueous polar solvent, active compound, optionally flavoring agent, and propellant; formulation C: non-polar solvent, active compound, and optional flavoring agent; and formulation D: non-polar solvent, active compound, optional flavoring agent, and propellant. Thus, a polar lingual spray contained albuterol sulfate 0.1-10, water 5-90, ethanol 1-10, sorbitol 0.1-5, aspartame 0.01-0.5, and flavors 0.1-5%.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L4 ANSWER 7 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2003:319257 CAPLUS
DOCUMENT NUMBER:	138:343856
TITLE:	Buccal sprays or capsules containing cardiovascular or renal drugs
INVENTOR(S):	Dugger, Harry A., III
PATENT ASSIGNEE(S):	USA
SOURCE:	U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S. Ser. No. 537,118. CODEN: USXXCO
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	19

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030077229	A1	20030424	US 2002-230075	20020829 <--
WO 9916417	A1	19990408	WO 1997-US17899	19971001 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
EP 1036561	A1	20000920	EP 2000-109357	19971001 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
EP 1952802	A2	20080806	EP 2007-23005	19971001
EP 1952802	A3	20090617		
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
EP 2042161	A1	20090401	EP 2008-20267	19971001
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
CA 2496769	A1	20040311	CA 2003-2496769	20030827
WO 2004019909	A2	20040311	WO 2003-US26853	20030827
WO 2004019909	A3	20040708		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003270014	A1	20040319	AU 2003-270014	20030827
EP 1536769	A2	20050608	EP 2003-751909	20030827
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
JP 2006502147	T	20060119	JP 2004-531569	20030827
US 20050025713	A1	20050203	US 2004-928979	20040827
US 20080170995	A1	20080717	US 2007-929368	20071030
JP 2009079060	A	20090416	JP 2008-266598	20081015
US 20090123387	A1	20090514	US 2009-351490	20090109
JP 2009149675	A	20090709	JP 2009-41207	20090224

PRIORITY APPLN. INFO.:		
	WO 1997-US17899	A2 19971001
	US 2000-537118	A2 20000329
	EP 1997-911621	A3 19971001
	JP 2000-513555	A3 19971001
	US 2002-230075	A 20020829
	WO 2003-US26853	W 20030827

AB Buccal aerosol sprays or capsules using polar and non-polar solvent have now been developed which provide biol. active compds. for rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise formulation A: aqueous polar solvent, active compound, and optional flavoring agent; formulation B: aqueous polar solvent, active compound, optionally flavoring agent, and propellant; formulation C: non-polar solvent, active compound, and optional flavoring agent; and formulation D: non-polar solvent, active compound, optional flavoring agent, and propellant. Thus, a polar lingual spray contained

isoproterenol-HCl 0.5-6, water 50-75, EtOH 5-10, PEG 5-15, sorbitol 0.4-1.0, aspartame 0.04-0.1, and flavors 2-3%.

L4 ANSWER 8 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2003:319256 CAPLUS
DOCUMENT NUMBER: 138:343855
TITLE: Buccal sprays or capsules containing drugs for treating endocrine disorders
INVENTOR(S): Dugger, Harry A., III
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 12 pp., Cont.-in-part of U.S. Ser. No. 537,118.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 19
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20030077228	A1	20030424	US 2002-230073	20020829 <--
WO 9916417	A1	19990408	WO 1997-US17899	19971001 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW				
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
EP 1036561	A1	20000920	EP 2000-109357	19971001 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
EP 1952802	A2	20080806	EP 2007-23005	19971001
EP 1952802	A3	20090617		
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
EP 2042161	A1	20090401	EP 2008-20267	19971001
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
WO 2004019911	A2	20040311	WO 2003-US26857	20030827
WO 2004019911	A3	20040715		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003270017	A1	20040319	AU 2003-270017	20030827
US 20050180923	A1	20050818	US 2003-671708	20030929
US 20050025715	A1	20050203	US 2004-928995	20040827
US 20060210484	A1	20060921	US 2006-440095	20060525
JP 2009079060	A	20090416	JP 2008-266598	20081015
JP 2009149675	A	20090709	JP 2009-41207	20090224
PRIORITY APPLN. INFO.:			WO 1997-US17899	A2 19971001
			US 2000-537118	A2 20000329
			EP 1997-911621	A3 19971001
			JP 2000-513555	A3 19971001

US 2002-230073 A 20020829
WO 2003-US26857 W 20030827
US 2003-671708 A3 20030929

AB Buccal aerosol sprays or capsules using polar and non-polar solvent have now been developed which provide biol. active compds. for rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprise formulation A: aqueous polar solvent, active compound, and optional flavoring agent; formulation B: aqueous polar solvent, active compound, optionally flavoring agent, and propellant; formulation C: non-polar solvent, active compound, and optional flavoring agent; and formulation D: non-polar solvent, active compound, optional flavoring agent, and propellant. Thus, a polar solvent formulation contained glyburide 0.6-10, EtOH 70-97, water 0.2-2, flavors 0.1-2.5, and propellant 3-4%.

L4 ANSWER 9 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:975101 CAPLUS
DOCUMENT NUMBER: 138:232059
TITLE: Inhibitory interactions between 5-HT3 and P2X channels in submucosal neurons
AUTHOR(S): Barajas-Lopez, Carlos; Montano, Luis M.; Espinosa-Luna, Rosa
CORPORATE SOURCE: Department of Anatomy and Cell Biology, Queen's University, Kingston, ON, K7L 3N6, Can.
SOURCE: American Journal of Physiology (2002), 283(6, Pt. 1), G1238-G1248
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Inhibitory interactions between 5-HT subtype 3 (5-HT3) and P2X receptors were characterized using whole cell recording techniques. Currents induced by 5-HT (I5-HT) and ATP (IATP) were blocked by tropisetron (or ondansetron) and pyridoxalphosphate-6-azophenyl-2',4'-disulfonic acid, resp. Currents induced by 5-HT + ATP (I5-HT+ATP) were only as large as the current induced by the most effective transmitter, revealing current occlusion. Occlusion was observed at membrane potentials of -60 and 0 mV (for inward currents), but it was not present at +40 mV (for outward currents). Kinetic and pharmacol. properties of I5-HT+ATP indicate that they are carried through 5-HT3 and P2X channels. Current occlusion occurred as fast as activation of I5-HT and IATP, was still present in the absence of Ca2+ or Mg2+, after adding staurosporine, genistein, K-252a, or N-ethylmaleimide to the pipet solution, after substituting ATP with α,β-methylene ATP or GTP with GTP-γ-S in the pipet, and was observed at 35°, 23°, and 8°. These results are in agreement with a model that considers that 5-HT3 and P2X channels are in functional clusters and that these channels might directly inhibit each other.

OS.CITING REF COUNT: 25 THERE ARE 25 CAPLUS RECORDS THAT CITE THIS RECORD (25 CITINGS)
REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 10 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:833515 CAPLUS
DOCUMENT NUMBER: 137:333176
TITLE: As-needed administration of tricyclic and other non-SRI antidepressant drugs to treat premature ejaculation
INVENTOR(S): Tam, Peter; Gesundheit, Neil; Wilson, Leland F.
PATENT ASSIGNEE(S): Vivus, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 15 pp., Cont.-in-part of U.S.

Ser. No. 721,412.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20020161016	A1	20021031	US 2001-996407	20011121 <--
US 6946141	B2	20050920		
US 6495154	B1	20021217	US 2000-721412	20001121 <--

PRIORITY APPLN. INFO.: US 2000-721412 A2 20001121

AB A method is provided for treatment of premature ejaculation by administration of an antidepressant drug selected from tricyclic antidepressants, tetracyclic antidepressants, MAO inhibitors, azaspirone antidepressants, and atypical non-SRI antidepressants. In a preferred embodiment, administration is on as "as-needed" basis, i.e., the drug is administered immediately or at most several hours prior to sexual activity. Pharmaceutical formulations and packaged kits are also provided.

L4 ANSWER 11 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:717534 CAPLUS
DOCUMENT NUMBER: 138:11708
TITLE: Cosensitivity of vagal mucosal afferents to histamine and 5-HT in the rat jejunum
AUTHOR(S): Kreis, M. E.; Jiang, W.; Kirkup, A. J.; Grundy, D.
CORPORATE SOURCE: Department of General Surgery, University Hospital Tubingen, Tubingen, D-72076, Germany
SOURCE: American Journal of Physiology (2002), 283(3, Pt. 1), G612-G617
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A complex sensitivity of afferent nerves in the mesentery of the rat jejunum to systemic administration of histamine has recently been demonstrated. In the present study, the authors aimed to characterize sub-populations of mesenteric afferents that mediate this afferent nerve response. Multiunit afferent discharge was recorded from mesenteric nerves supplying the proximal jejunum in anesthetized rats. The majority of mesenteric bundles (84%) exhibited biphasic responses to histamine (8 μmol/kg), and these bundles also responded to 2-methyl-5-HT (2m5HT). In contrast, monophasic responses lacked a short-latency component, and these bundles failed to respond to 2m5HT. Single-unit anal. revealed a population of afferents that possessed cosensitivity for 2m5HT and histamine. This population of afferents was absent in chronically vagotomized animals, whereas mucosal anesthesia with luminal lidocaine reversibly converted the biphasic profile to a monophasic one. Ondansetron (500 μg/kg) blocked the response to 2m5HT with no effect on the profile of the histamine response, whereas pyrilamine (5 mg/kg) blocked the histamine response without affecting the response to 2m5HT. The authors conclude that histamine-sensitive afferents exist in the rat proximal jejunum that also respond to 5-HT via the 5-HT3 receptor. These fibers appear to be vagal afferents originating in the intestinal mucosa and may be involved in the organization of mast cell-mediated responses.

OS.CITING REF COUNT: 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
REFERENCE COUNT: 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 12 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2002:241329 CAPLUS
DOCUMENT NUMBER: 136:284433
TITLE: Administration of phosphodiesterase inhibitors for the treatment of premature ejaculation
INVENTOR(S): Wilson, Leland F.; Doherty, Paul C.; Place, Virgil A.; Smith, William L.; Abdel-Hamid, Abdou Ali Ibrahim Aboubakr
PATENT ASSIGNEE(S): Vivus, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 21 pp., Cont.-in-part of U.S. Ser. No. 467,094.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 20020037828	A1	20020328	US 2001-888250	20010621	<--
US 6403597	B2	20020611			
US 6037346	A	20000314	US 1998-181070	19981027	<--
US 6548490	B1	20030415	US 1999-467094	19991210	<--
CA 2451152	A1	20030103	CA 2002-2451152	20020325	<--
WO 2003000343	A2	20030103	WO 2002-US9415	20020325	<--
WO 2003000343	A3	20040325			
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZM, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2002248712	A1	20030108	AU 2002-248712	20020325	<--
EP 1418896	A2	20040519	EP 2002-717729	20020325	
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2005519851	T	20050707	JP 2003-506984	20020325	
AU 2005248938	A1	20060202	AU 2005-248938	20051223	

PRIORITY APPLN. INFO.:

US 1997-958816	B2	19971028
US 1998-181070	A2	19981027
US 1999-467094	A2	19991210
AU 2001-22566	A3	20001208
US 2001-888250	A	20010621
WO 2002-US9415	W	20020325

AB A method is provided for treatment of premature ejaculation by administration of a phosphodiesterase inhibitor, e.g., an inhibitor of a Type III, Type IV, or Type V phosphodiesterase. In a preferred embodiment, administration is on as "as needed" basis, i.e., the drug is administered immediately or several hours prior to sexual activity. Pharmaceutical formulations and packaged kits are also provided. Zaprinast 1.0, mannitol 1.0, microcryst. cellulose 2.0, and magnesium stearate 10 mg are blended in a suitable mixer and then compressed into sublingual tablets. Each sublingual tablet contains 10 mg zaprinast.
OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L4 ANSWER 13 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:39607 CAPLUS
DOCUMENT NUMBER: 136:96093
TITLE: Methods and compositions using a sibutramine metabolite or other dopamine uptake inhibitors for the treatment and prevention of sexual dysfunction
INVENTOR(S): Jerussi, Thomas P.; Senanayake, Chrisantha H.; Fang, Qun K.
PATENT ASSIGNEE(S): Sepracor, Inc., USA
SOURCE: U.S., 21 pp., Cont.-in-part of U.S. Ser. No. 372,158.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 6339106	B1	20020115	US 2000-662135	20000914	<--
US 6331571	B1	20011218	US 1999-372158	19990811	<--
EP 1475086	A2	20041110	EP 2004-18454	19990823	
EP 1475086	A3	20061213			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL					
US 20020010198	A1	20020124	US 2001-770663	20010129	<--
US 6476078	B2	20021105			
CA 2422246	A1	20020321	CA 2001-2422246	20010913	<--
WO 2002022114	A2	20020321	WO 2001-US28598	20010913	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
AU 2001089062	A	20020326	AU 2001-89062	20010913	<--
EP 1320360	A1	20030625	EP 2001-968848	20010913	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2004529850	T	20040930	JP 2002-526365	20010913	
AU 2001289062	B2	20070329	AU 2001-289062	20010913	
US 20030096792	A1	20030522	US 2002-278097	20021023	<--
US 6974837	B2	20051213			
US 20030195261	A1	20031016	US 2003-395298	20030325	<--
US 7071234	B2	20060704			
US 20040067957	A1	20040408	US 2003-665448	20030922	
US 20040092481	A1	20040513	US 2003-693980	20031028	
US 20040116534	A1	20040617	US 2003-717653	20031121	
US 20040162355	A1	20040819	US 2004-769860	20040203	
AU 2004200875	A1	20040401	AU 2004-200875	20040303	
AU 2004200875	B2	20061026			
RU 2358719	C2	20090620	RU 2004-116282	20040527	
AU 2007200334	A1	20070215	AU 2007-200334	20070125	

PRIORITY APPLN. INFO.:

US 1999-372158	A2	19990811
US 1998-97665P	P	19980824
US 1998-99306P	P	19980902
AU 1999-57817	A3	19990823
EP 1999-945137	A3	19990823
RU 2001-107831	A3	19990823
US 2000-662135	A2	20000914
US 2001-770663	A3	20010129

	WO 2001-US28598	W	20010913
	US 2001-806	A3	20011204
	US 2002-278097	A3	20021023
	AU 2004-200875	A3	20040303

AB Methods are disclosed for the treatment and prevention of sexual dysfunction. The methods comprise the administration of a dopamine reuptake inhibitor and optionally an addnl. pharmacol. active compound Pharmaceutical compns. and dosage forms are also disclosed that comprise a dopamine reuptake inhibitor and optionally an addnl. pharmacol. active compound Preferred dopamine reuptake inhibitors are racemic or optically pure sibutramine metabolites and pharmaceutically acceptable salts, solvates, and clathrates thereof. Preferred addnl. pharmacol. active compds. include drugs that affect the central nervous system, such as 5-HT3 antagonists. Preparation of sibutramine metabolites is described.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 125 THERE ARE 125 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 14 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2002:23847 CAPLUS

DOCUMENT NUMBER: 136:79797

TITLE: Bupropion metabolites, and preparation thereof, for treatment of sexual dysfunction

INVENTOR(S): Fang, Qun Kevin; Senanayake, Chrisantha Hugh; Grover, Paul

PATENT ASSIGNEE(S): Sepracor, Inc., USA

SOURCE: U.S., 26 pp., Cont.-in-part of U.S. 510,241. CODEN: USXXAM

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 3

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6337328	B1	20020108	US 2000-640725	20000818 <--
US 6342496	B1	20020129	US 2000-510241	20000222 <--
EP 1759701	A2	20070307	EP 2006-120882	20000229
EP 1759701	A3	20070314		
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, AL, BA, HR, MK, YU				
CA 2400482	A1	20010830	CA 2000-2400482	20000823 <--
WO 2001062257	A2	20010830	WO 2000-US23080	20000823 <--
WO 2001062257	A3	20020704		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1259243	A2	20021127	EP 2000-957684	20000823 <--
EP 1259243	B1	20060628		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
HU 2003000030	A2	20030528	HU 2003-30	20000823 <--
JP 2003529563	T	20031007	JP 2001-561322	20000823 <--
AU 2000269268	B2	20050908	AU 2000-269268	20000823

EP 1602369	A2	20051207	EP 2005-106426		20000823	
EP 1602369	A3	20070214				
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL						
AT 331520	T	20060715	AT 2000-957684		20000823	
ES 2261234	T3	20061116	ES 2000-957684		20000823	
US 20020052340	A1	20020502	US 2001-987930		20011116	<--
US 20020052341	A1	20020502	US 2001-987931		20011116	<--
MX 2002008093	A	20030523	MX 2002-8093		20020820	<--
US 20060058300	A1	20060316	US 2005-253689		20051020	
AU 2005247034	A1	20060119	AU 2005-247034		20051222	
PRIORITY APPLN. INFO.:			US 1999-122277P	P	19990301	
			US 1999-148324P	P	19990811	
			US 2000-510241	A2	20000222	
			US 2000-510241P	P	20000222	
			EP 2000-913649	A3	20000229	
			US 2000-640725	A	20000818	
			AU 2000-69268	A3	20000823	
			EP 2000-957684	A3	20000823	
			WO 2000-US23080	W	20000823	
			US 2001-987931	A3	20011116	

AB Methods are disclosed which use metabolites of bupropion (preparation described) for treating sexual dysfunction. Tablet formulations are included.

OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (14 CITINGS)

REFERENCE COUNT: 143 THERE ARE 143 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 15 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2001:798099 CAPLUS

DOCUMENT NUMBER: 135:348894

TITLE: Drug delivery device for insertion in the vagina, rectum or nasal cavity

INVENTOR(S): Knox, Peter

PATENT ASSIGNEE(S): Metris Therapeutics Limited, UK

SOURCE: PCT Int. Appl., 32 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001080937	A1	20011101	WO 2001-GB1789	20010420 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2376791	A1	20011101	CA 2001-2376791	20010420 <--
CA 2376791	C	20081125		
GB 2364916	A	20020213	GB 2001-9768	20010420 <--
GB 2364916	B	20020731		
US 20020022816	A1	20020221	US 2001-840004	20010420 <--
US 6758840	B2	20040706		

EP 1200151 A1 20020502 EP 2001-921653 20010420 <--
EP 1200151 B1 20041013
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
JP 2003531182 T 20031021 JP 2001-578030 20010420 <--
AU 776434 B2 20040909 AU 2001-48621 20010420
AT 279230 T 20041015 AT 2001-921653 20010420
HK 1045952 A1 20050401 HK 2002-107097 20020926
PRIORITY APPLN. INFO.: GB 2000-9914 A 20000420
WO 2001-GB1789 W 20010420

AB The invention relates to drug delivery devices for insertion into the vagina, rectum or nasal cavity comprising a body, a layer of fluid-impermeable material on at least part of said body and one or more pharmaceutical agents disposed on the surface of the material remote from said body, wherein said body comprises absorbent material. The devices exploit the highly vascularized nature of the vaginal, nasal and rectal mucosal tissue to deliver pharmaceutical agents to localized areas and/or into underlying tissues. A fluid-impermeable material is any one of polyethylene, polypropylene, a polyester, a polyolefin, a rubber such as a polybutadiene and a butadiene-styrene rubber or siliconized materials (thickness of 10 μm to 2 mm). The fluid-impermeable material is applied to the surface of the device in the form of one or more discrete patches, and pharmaceutical agent is disposed on the device in aliquots that are coincident in position with said patches of fluid-impermeable material. The patches of said fluid-impermeable material are in the form of circles, rectangles, squares, triangles, ellipses or circumferential rings. The amount of pharmaceutical agent, such as antifibrinolytics, antiinflammatory agents, tocolytic agents, antiemetics, antimigraine agents, bronchodilators, or diuretics disposed on the surface is between 100 μg and 10 mg. For example, a layer of methacrylate polymer, obtained from com. available adhesives, was formed on the surface of three com. available tampons by applying thin layers of unpolymd. material to small areas of the tampon surface and allowing the layers to set hard in an oven at 120°. About 20 μL of silver nitrate solution was applied to the surface of the polymer layers of each tampon. Following drying, a tissue and gauze layer that had been soaked in sodium hydroxide was applied to the surface of each tampon. These tissue layers were intended to model the surface of the vaginal mucosa. The ensuing reaction between the silver nitrate and the sodium hydroxide caused insol. oxides of silver to be deposited on each tissue and gave a visual indication of the amount of silver nitrate that had been available at the surface of each tampon. A photograph of the tissue layers that were obtained following application to the surface of three sep. tampons showed that there was more silver nitrate available for reaction with the sodium hydroxide in the tissue in the areas where there was a layer of methacrylate polymer that acted as a fluid-impermeable layer. In contrast, in the areas where there was no methacrylate polymer layer, much of the silver nitrate had been absorbed or diffused into the body of the tampon and was no longer available for reaction with the sodium hydroxide in the tissue. Consequently, the presence of a fluid-impermeable layer increases the concentration of silver nitrate available for reaction.

OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 16 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:750727 CAPLUS
DOCUMENT NUMBER: 136:15549
TITLE: Intestinal serotonin acts as paracrine substance to mediate pancreatic secretion stimulated by luminal factors

AUTHOR(S): Li, Y.; Wu, X. Y.; Zhu, J. X.; Owyang, C.
CORPORATE SOURCE: Gastroenterology Research Unit, Department of Internal Medicine, University of Michigan Health System, Ann Arbor, MI, 48109-0682, USA
SOURCE: American Journal of Physiology (2001), 281(4, Pt. 1), G916-G923
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors recently demonstrated that luminal factors such as osmolality, disaccharides, and mech. stimulation evoke pancreatic secretion by activating 5-hydroxytryptamine subtype 3 (serotonin-3, 5-HT3) receptors on mucosal vagal afferent fibers in the intestine. The authors hypothesized that 5-HT released by luminal stimuli acts as a paracrine substance, activating the mucosal vagal afferent fibers to stimulate pancreatic secretion. In the in vivo rat model, luminal perfusion of maltose or hypertonic NaCl increased 5-HT level threefold in intestinal effluent perfusates. Similar levels were observed after intraluminal 10-5 M 5-HT perfusion. These treatments did not affect 5-HT blood levels. In a sep. study, intraduodenal, but not intraileal, 5-HT application induced a dose-dependent increase in pancreatic protein secretion, which was not blocked by the CCK-A antagonist CR-1409. Acute vagotomy, methscopolamine, or perivagal or intestinal mucosal application of capsaicin abolished 5-HT-induced pancreatic secretion. In conscious rats, luminal 10-5 M 5-HT administration produced a 90% increase in pancreatic protein output, which was markedly inhibited by the 5-HT3 antagonist ondansetron. In conclusion, luminal stimuli induce 5-HT release, which in turn activates 5-HT3 receptors on mucosal vagal afferent terminals. In this manner, 5-HT acts as a paracrine substance to stimulate pancreatic secretion via a vagal cholinergic pathway.

OS.CITING REF COUNT: 37 THERE ARE 37 CAPLUS RECORDS THAT CITE THIS RECORD (37 CITINGS)
REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 17 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2001:730530 CAPLUS
DOCUMENT NUMBER: 135:293950
TITLE: A self-emulsifying system combined with a polymer matrix for transmucosal and transdermal delivery
INVENTOR(S): Hong, Chung Il; Shin, Hee Jong; Ki, Min Hyo; Lee, Seok Kyu; Kweon, Don Sun
PATENT ASSIGNEE(S): Chong Kun Dang Pharmaceutical Corp., S. Korea
SOURCE: PCT Int. Appl., 41 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001072282	A1	20011004	WO 2001-KR509	20010329 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

KR 2001093728 A 20011029 KR 2001-16140 20010328 <--
US 20030129219 A1 20030710 US 2002-239529 20020923 <--
PRIORITY APPLN. INFO.: KR 2000-16257 A 20000329
WO 2001-KR509 W 20010329

AB A novel pharmaceutical composition of a self-emulsifying matrix preparation, which is a preparation for transmucosal or transdermal absorption in which a self-emulsifying drug delivery system is grafted to a polymeric matrix preparation is described. For this, fatty alc., fatty acid or their derivs. of 6 to 20 carbon atoms having a drug absorption-accelerating action through the skin or mucous membrane is used as an oil phase. Also, to increase the drug content in the matrix, a liquid phase material having a b.p. of 100°C or more is used as a solution adjuvant. Using such materials, the self-emulsifying system with a surfactant is prepared A hydrophilic or hydrophobic polymer is added and dissolved in the self-emulsifying system, and the resulting mixture is dried to prepare the matrix preparation containing the self-emulsifying system. The self-emulsifying matrix preparation thus prepared maintains a constant drug-releasing rate during its application period by virtue of its excellent stability and exhibits an extraordinarily high skin-absorption rate. For example, a self-emulsifying system was prepared using oleyl alc. 10, glycerin (1) oleic acid ester 10, diethylene glycol monoethyl ether 40, and Cremophor RH40 40 parts, resp., as an oily phase. Upon the addition of water, a self-emulsification was obtained. To 10 g of the self-emulsifying matrix prepared was added 5 g of arecoline monohydrobromide as a drug. Sixty grams of poly(ethylene oxide) was dissolved into 30 g of water and 30 g of ethanol to form a polymer solution This prepolymer solution was added to the self-emulsifying system containing the drug to give a transparent viscous solution, which was then dried at 80° for 10 min to form a self-emulsifying matrix with a thickness of 505 μm. During the process of drying, UV ray may be irradiated for 5 min, if necessary.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 18 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2000:725436 CAPLUS
DOCUMENT NUMBER: 133:301171
TITLE: Compositions and methods for improved delivery of ionizable hydrophobic therapeutic agents
INVENTOR(S): Chen, Feng-jing; Patel, Manesh V.
PATENT ASSIGNEE(S): Lipocine, Inc., USA
SOURCE: PCT Int. Appl., 99 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000059475	A1	20001012	WO 2000-US7342	20000316 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,

DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6383471	B1	20020507	US 1999-287043	19990406	<--
CA 2366702	A1	20001012	CA 2000-2366702	20000316	<--
CA 2366702	C	20090526			
EP 1165048	A1	20020102	EP 2000-916547	20000316	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.:	US 1999-287043	A	19990406
	WO 2000-US7342	W	20000316

AB The present invention is directed to a pharmaceutical composition including a hydrophobic therapeutic agent having at least one ionizable functional group, and a carrier. The carrier includes an ionizing agent capable of ionizing the functional group, a surfactant, and optionally solubilizers, triglycerides, and neutralizing agents. The invention further relates to a method of preparing such compns. by providing a composition of an ionizable hydrophobic therapeutic agent, an ionizing agent, and a surfactant, and neutralizing a portion of the ionizing agent with a neutralizing agent. The compns. of the invention are particularly suitable for use in oral dosage forms. A carrier containing concentrated phosphoric acid 0.025, Tween-20 0.3, Arlacel 186 0.2, sodium taurocholate 0.15, propylene glycol 0.3 g was formulated. Itraconazole was included in the carrier at 30 mg/mL for testing the stability of the itraconazole solution upon dilution in simulated gastric fluid.

OS.CITING REF COUNT:	43	THERE ARE 43 CAPLUS RECORDS THAT CITE THIS RECORD (43 CITINGS)
REFERENCE COUNT:	3	THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 19 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER:	2000:627938 CAPLUS
DOCUMENT NUMBER:	133:227784
TITLE:	Bupropion metabolites and methods of their synthesis and therapeutic uses and compositions
INVENTOR(S):	Jerussi, Thomas P.; McCullough, John R.; Senanayake, Chrisantha H.; Fang, Qun K.
PATENT ASSIGNEE(S):	Sepracor Inc., USA
SOURCE:	PCT Int. Appl., 41 pp. CODEN: PIXXD2
DOCUMENT TYPE:	Patent
LANGUAGE:	English
FAMILY ACC. NUM. COUNT:	3
PATENT INFORMATION:	

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 2000051546	A2	20000908	WO 2000-US5109	20000229	<--
WO 2000051546	A3	20010111			

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

CA 2362361	A1	20000908	CA 2000-2362361	20000229	<--
AU 2000035055	A	20000921	AU 2000-35055	20000229	<--
AU 775642	B2	20040812			
JP 2004513061	T	20040430	JP 2000-602018	20000229	
EP 1759701	A2	20070307	EP 2006-120882	20000229	

EP 1759701 A3 20070314
R: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE, AL, BA, HR, MK, YU
AU 2005247034 A1 20060119 AU 2005-247034 20051222
PRIORITY APPLN. INFO.: US 1999-122277P P 19990301
US 1999-148324P P 19990811
US 2000-510241 A 20000222
US 2000-510241P P 20000222
EP 2000-913649 A3 20000229
WO 2000-US5109 W 20000229
AU 2000-69268 A3 20000823
OTHER SOURCE(S): MARPAT 133:227784
AB Methods and compns. are disclosed which utilize metabolites of bupropion for treating disorders ameliorated by inhibition of neuronal monoamine reuptake. Such disorders include, but are not limited to, erectile dysfunction, affective disorders, cerebral function disorders, cigarette smoking, and incontinence. The invention further discloses methods of making optically pure bupropion metabolites.
OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

L4 ANSWER 20 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2000:296267 CAPLUS
DOCUMENT NUMBER: 133:27069
TITLE: Activation of intrinsic afferent pathways in submucosal ganglia of the guinea pig small intestine
AUTHOR(S): Pan, Hui; Gershon, Michael D.
CORPORATE SOURCE: Department of Anatomy and Cell Biology, Columbia University College of Physicians and Surgeons, New York, NY, 10032, USA
SOURCE: Journal of Neuroscience (2000), 20(9), 3295-3309
CODEN: JNRSDS; ISSN: 0270-6474
PUBLISHER: Society for Neuroscience
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The enteric nervous system contains intrinsic primary afferent neurons that allow mucosal stimulation to initiate reflexes without CNS input. We tested the hypothesis that submucosal primary afferent neurons are activated by 5-hydroxytryptamine (5-HT) released from the stimulated mucosa. Fast and/or slow EPSPs were recorded in submucosal neurons after the delivery of exogenous 5-HT, WAY100325 (a 5-HT1P agonist), mech., or elec. stimuli to the mucosa of myenteric plexus-free prepns. (± extrinsic denervation). These events were responses of second-order cells to transmitters released by excited primary afferent neurons. After all stimuli, fast and slow EPSPs were abolished by a 5-HT1P antagonist, N-acetyl-5-hydroxytryptophyl-5-hydroxytryptophan amide, and by 1.0 μM tropisetron, but not by 5-HT4-selective antagonists (SB204070 and GR113808A) or 5-HT3-selective antagonists (ondansetron and 0.3 μM tropisetron). Fast EPSPs in second-order neurons were blocked by hexamethonium, and most slow EPSPs were blocked by an antagonist of human calcitonin gene-related peptide (hCGRP8-37). HCGRP8-37 also inhibited the spread of excitation in the submucosal plexus, assessed by measuring the uptake of FM2-10 and induction of c-fos. In summary, data are consistent with the hypothesis that 5-HT from enterochromaffin cells in response to mucosal stimuli initiates reflexes by stimulating 5-HT1P receptors on submucosal primary afferent neurons. Second-order neurons respond to these cholinergic/CGRP-containing cells with nicotinic fast EPSPs and/or CGRP-mediated slow EPSPs. Slow EPSPs are necessary for excitation to spread within the submucosal plexus. Because some

second-order neurons contain also CGRP, primary afferent neurons may be multifunctional and also serve as interneurons.

OS.CITING REF COUNT: 84 THERE ARE 84 CAPLUS RECORDS THAT CITE THIS RECORD (84 CITINGS)

REFERENCE COUNT: 78 THERE ARE 78 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 21 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 2000:144721 CAPLUS

DOCUMENT NUMBER: 132:189679

TITLE: Methods of using and compositions comprising dopamine reuptake inhibitors

INVENTOR(S): Jerussi, Thomas P.; Senanayake, Chrisantha H.; Fang, Qun K.

PATENT ASSIGNEE(S): Sepracor Inc., USA

SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 5

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 2000010551	A2	20000302	WO 1999-US19167	19990823	<--
WO 2000010551	A3	20000921			
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA, ZW					
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
US 6331571	B1	20011218	US 1999-372158	19990811	<--
CA 2341441	A1	20000302	CA 1999-2341441	19990823	<--
AU 9957817	A	20000314	AU 1999-57817	19990823	<--
AU 772303	B2	20040422			
EP 1107746	A2	20010620	EP 1999-945137	19990823	<--
EP 1107746	B1	20041013			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
BR 9913325	A	20011002	BR 1999-13325	19990823	<--
HU 2001003408	A2	20020529	HU 2001-3408	19990823	<--
HU 2001003408	A3	20021028			
JP 2002523366	T	20020730	JP 2000-565873	19990823	<--
NZ 510193	A	20030926	NZ 1999-510193	19990823	<--
AT 279184	T	20041015	AT 1999-945137	19990823	
RU 2238084	C2	20041020	RU 2001-107831	19990823	
EP 1475086	A2	20041110	EP 2004-18454	19990823	
EP 1475086	A3	20061213			
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL					
ES 2226435	T3	20050316	ES 1999-945137	19990823	
CN 1735407	A	20060215	CN 1999-812466	19990823	
CN 100415222	C	20080903			
ZA 2001001498	A	20020222	ZA 2001-1498	20010222	<--
NO 2001000943	A	20010423	NO 2001-943	20010223	<--
IN 2001CN00405	A	20050304	IN 2001-CN405	20010322	
US 20020188029	A1	20021212	US 2001-806	20011204	<--
US 6538034	B2	20030325			
US 20030195261	A1	20031016	US 2003-395298	20030325	<--

US 7071234	B2	20060704			
AU 2004200875	A1	20040401	AU 2004-200875		20040303
AU 2004200875	B2	20061026			
RU 2358719	C2	20090620	RU 2004-116282		20040527
KR 2006081725	A	20060713	KR 2006-712844		20060627
KR 887008	B1	20090304			
HK 1088238	A1	20090605	HK 2006-108671		20060804
AU 2007200334	A1	20070215	AU 2007-200334		20070125
KR 2008011354	A	20080201	KR 2008-701197		20080115
IN 2008CN02927	A	20090306	IN 2008-CN2927		20080611
PRIORITY APPLN. INFO.:			US 1998-97665P	P	19980824
			US 1998-99306P	P	19980902
			US 1999-372158	A	19990811
			AU 1999-57817	A3	19990823
			EP 1999-945137	A3	19990823
			RU 2001-107831	A3	19990823
			WO 1999-US19167	W	19990823
			KR 2001-702288	A3	20010223
			IN 2001-CN405	A3	20010322
			US 2001-806	A3	20011204
			AU 2004-200875	A3	20040303
			KR 2006-712844	A3	20060627

AB Methods are disclosed for the treatment and prevention of disorders and conditions including, but are not limited to, erectile dysfunction, affective disorders, weight gain, cerebral functional disorders, pain, obsessive-compulsive disorder, substance abuse, chronic disorders, anxiety, eating disorders, migraines, and incontinence. The methods comprise the administration of a dopamine reuptake inhibitor and optionally an addnl. pharmacol. active compound Pharmaceutical compns. and dosage forms are also disclosed that comprise a dopamine reuptake inhibitor and optionally an addnl. pharmacol. active compound Preferred dopamine reuptake inhibitors are racemic or optically pure sibutramine metabolites and pharmaceutically acceptable salts, solvates, and clathrates thereof. Preferred addnl. pharmacol. active compds. include drugs that affect the central nervous system, such as 5-HT3, antagonists.

OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)

REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 22 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1999:233778 CAPLUS

DOCUMENT NUMBER: 130:272007

TITLE: Buccal spray or capsule compositions containing polar and non-polar solvents for transmucosal administration of drugs

INVENTOR(S): Dugger, Harry A., III

PATENT ASSIGNEE(S): Flemington Pharmaceutical Corporation, USA

SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 19

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9916417	A1	19990408	WO 1997-US17899	19971001 <--

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US,

UZ, VN, YU, ZW
RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

CA 2306024 A1 19990408 CA 1997-2306024 19971001 <--
AU 9748946 A 19990423 AU 1997-48946 19971001 <--
EP 1019019 A1 20000719 EP 1997-911621 19971001 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
EP 1029536 A1 20000823 EP 2000-109347 19971001 <--
EP 1029536 B1 20071128
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
EP 1036561 A1 20000920 EP 2000-109357 19971001 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO
JP 2001517689 T 20011009 JP 2000-513555 19971001 <--
ES 2293875 T3 20080401 ES 2000-109347 19971001
EP 1952802 A2 20080806 EP 2007-23005 19971001
EP 1952802 A3 20090617
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
EP 2042161 A1 20090401 EP 2008-20267 19971001
R: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE
US 20030039680 A1 20030227 US 2002-100156 20020318 <--
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US 20030077227 A1 20030424 US 2002-230060 20020829 <--
US 20030077228 A1 20030424 US 2002-230073 20020829 <--
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US 20030082107 A1 20030501 US 2002-230080 20020829 <--
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US 20030095927 A1 20030522 US 2002-230086 20020829 <--
US 20030185761 A1 20031002 US 2002-230059 20020829 <--
US 20030190286 A1 20031009 US 2002-230072 20020829 <--
US 20030211047 A1 20031113 US 2002-327195 20021224 <--
US 6998110 B2 20060214
US 20040062716 A1 20040401 US 2003-663817 20030917
US 20040136913 A1 20040715 US 2003-671710 20030929
US 20040136914 A1 20040715 US 2003-671717 20030929
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US 20040141923 A1 20040722 US 2003-671720 20030929
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US 20050163719 A1 20050728 US 2003-671709 20030929
US 20050180923 A1 20050818 US 2003-671708 20030929
US 20040120895 A1 20040624 US 2003-726585 20031204
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US 20040120896 A1 20040624 US 2003-726625 20031204
US 6969508 B2 20051129
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US 20050281752 A1 20051222 US 2005-211488 20050826
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US 20050287075 A1 20051229 US 2005-211487 20050826
US 20060165604 A1 20060727 US 2006-366663 20060303

US	20060159624	A1	20060720	US 2006-384444	20060321
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US	20060198790	A1	20060907	US 2006-429953	20060509
US	20060210484	A1	20060921	US 2006-440095	20060525
US	20060222597	A1	20061005	US 2006-442137	20060530
US	20060216240	A1	20060928	US 2006-443253	20060531
US	20060216241	A1	20060928	US 2006-443254	20060531
US	20070048229	A1	20070301	US 2006-443260	20060531
US	20080170995	A1	20080717	US 2007-929368	20071030
JP	2009079060	A	20090416	JP 2008-266598	20081015
US	20090118170	A1	20090507	US 2009-350898	20090108
US	20090131514	A1	20090521	US 2009-350915	20090108
US	20090162297	A1	20090625	US 2009-350602	20090108
US	20090123387	A1	20090514	US 2009-351490	20090109
US	20090124554	A1	20090514	US 2009-351606	20090109
US	20090162298	A1	20090625	US 2009-351576	20090109
US	20090186035	A1	20090723	US 2009-351179	20090109
US	20090186099	A1	20090723	US 2009-351275	20090109
JP	2009149675	A	20090709	JP 2009-41207	20090224
US	20090162300	A1	20090625	US 2009-394903	20090227

PRIORITY APPLN. INFO.:

EP	1997-911621	A3 19971001
EP	2000-109347	A3 19971001
JP	2000-513555	A3 19971001
WO	1997-US17899	A 19971001
US	2000-537118	A3 20000329
US	2002-100156	A1 20020318
US	2002-230059	A2 20020829
US	2002-230060	A2 20020829
US	2002-230072	A3 20020829
US	2002-230073	A2 20020829
US	2002-230075	A3 20020829
US	2002-230080	A3 20020829
US	2002-230084	A3 20020829
US	2002-230085	A2 20020829
US	2002-230086	A3 20020829
US	2002-327195	A1 20021224
US	2003-663817	B1 20030917
US	2003-671708	A3 20030929
US	2003-671709	A3 20030929
US	2003-671710	A3 20030929
US	2003-671715	A3 20030929
US	2003-671717	A3 20030929
US	2003-671719	A3 20030929
US	2003-671720	A3 20030929
US	2003-726585	A1 20031204
US	2003-726625	A1 20031204
US	2004-834815	A3 20040427
US	2006-366663	B1 20060303
US	2006-391297	B1 20060329
US	2006-429953	B1 20060509
US	2006-442137	B1 20060530

AB Buccal aerosol sprays or capsules containing biol. active peptides, CNS active amines, sulfonyl ureas, antibiotics, antifungals, antivirals, sleep inducers, antiasthmatics, bronchodilators, antiemetics, etc., are developed which provide rapid absorption through the oral mucosa, resulting in fast onset of effect. The buccal polar compns. of the invention comprises formulation: aqueous polar solvent 30-99.89%, active compound 0.001-60%, and optionally flavoring agent 0.1-10%. The non-polar composition of the invention comprises formulation: non-polar solvent 20-85%, active compound 0.005-50%, optionally flavoring agent 0.1-10%, and propellant 50-80%. A non-polar lingual spray composition contained zidovudine 25-35, soya oil 30-40, butane 60-70, and flavors 2-3 parts. resp.

OS.CITING REF COUNT: 13 THERE ARE 13 CAPLUS RECORDS THAT CITE THIS RECORD (17 CITINGS)
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 23 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1998:616734 CAPLUS
DOCUMENT NUMBER: 130:10883
TITLE: 5-HT induces cAMP production in crypt colonocytes at a 5-HT4 receptor
AUTHOR(S): Albuquerque, Francisco C., Jr.; Smith, Elise H.; Kellum, John M.
CORPORATE SOURCE: Department of Surgery, Medical College of Virginia/VCU, Richmond, VA, 23298-0161, USA
SOURCE: Journal of Surgical Research (1998), 77(2), 137-140
CODEN: JSGRA2; ISSN: 0022-4804
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Previous studies demonstrate that both 5-hydroxytryptamine (5-HT) and cAMP induce chloride efflux from crypt colonocytes in the rat distal colon; antagonist studies suggest that the 5-HT response is mediated primarily by the 5-HT4 receptor. Since this receptor is known to be pos. coupled to adenylate cyclase, the authors postulated that 5-HT should induce generation of cAMP, which should be inhibited by 5-HT4 antagonists. Mucosal cells from rat distal colon were taken by a sequential calcium chelation technique for enrichment of crypt cells. Cytokeratin stains demonstrated that >99% of cells were colonocytes. [3H]Thymidine uptake studies demonstrate a fivefold increased incorporation in this cell preparation compared to earlier fractions. 3-Isobutyl-1-methylxanthine (IBMX, 100 μM) was added to all cell suspensions to prevent cAMP metabolism Cell suspensions were incubated for 2 min at 37° with different concns. of 5-HT. The cAMP was measured by enzyme immunoassay. In another series of expts., 5-HT (0.3 μM) stimulation of cAMP was similarly measured in the presence and absence of 5-HT receptor antagonists: 10 μM 5-HTP-DP (5-HT1P), 0.1 μM ketanserin (5-HT2A), 0.3 μM ondansetron (5-HT3), 3 μM tropisetron (5-HT3 and 5-HT4), and 10 nM GR-113808 (5-HT4). 5-HT produced a dose-dependent increase in cAMP. The increase was significant at concns. ≥3 μM when compared to cells incubated with IBMX alone. In the second series of experiment, 5-HT-induced generation of cAMP at a dose of 0.3 μM was significantly inhibited in the presence of GR-113808 and tropisetron. 5-HT acts at a 5-HT4 receptor to induce production of cAMP in rat distal crypt colonocytes. (c) 1998 Academic Press.
OS.CITING REF COUNT: 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
REFERENCE COUNT: 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 24 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1998:586261 CAPLUS
DOCUMENT NUMBER: 129:281039
ORIGINAL REFERENCE NO.: 129:57207a,57210a
TITLE: Rectal preparations of serotonin receptor antagonists containing glycerides
INVENTOR(S): Hirano, Takahiko; Kozue, Masayoshi
PATENT ASSIGNEE(S): Hisamitsu Pharmaceutical Co., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 7 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese

FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 10236980	A	19980908	JP 1997-58322	19970226 <--
JP 4162735	B2	20081008		
PRIORITY APPLN. INFO.:			JP 1997-58322	19970226

AB The prepns., e.g. suppositories, ointments, creams, gels, etc., contain serotonin receptor antagonists and C8-18 (un)saturated fatty acid glycerin esters as base components. The glycerides preferably show OH value 50-90. The prepns. show good mucosal absorption and low irritation, and are useful for treatment of nausea and vomiting due to antitumor agents, irritable bowel syndrome, etc. A suppository was formulated from 98.0% Witepsol S 55 and 2.0% granisetron hydrochloride.

OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L4 ANSWER 25 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:751666 CAPLUS
DOCUMENT NUMBER: 128:57647
ORIGINAL REFERENCE NO.: 128:11151a,11154a
TITLE: Evidence for a 5-HT3 receptor involvement in the facilitation of peristalsis on mucosal application of 5-HT in the guinea pig isolated ileum
AUTHOR(S): Tuladhar, B. R.; Kaisar, M.; Naylor, R. J.
CORPORATE SOURCE: The School of Pharmacy, Postgraduate Studies in Pharmacology, University of Bradford, Bradford, BD7 1DP, UK
SOURCE: British Journal of Pharmacology (1997), 122(6), 1174-1178
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Stockton Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The 5-HT receptor involved in the effect of mucosal application of 5-HT to facilitate peristalsis was investigated in the isolated guinea pig ileum. An application of 5-HT (3-100 μM) to the mucosal surface (by inclusion of 5-HT in the Krebs-Henseleit solution passing through the lumen of the ileum) caused a concentration related facilitation of peristalsis characterized by a reduction in the peristaltic threshold. Peristalsis was not modified by methiothepin (0.1 μM), ritanserin (0.1 μM), ondansetron (5 μM), granisetron (1 μM) or SB 204070 (0.1 μM) administered alone to the mucosal surface. The concentration - response curve to mucosally applied 5-HT was not altered by the mucosally applied 5-HT1/2 receptor antagonist methiothepin (0.1 μM), the 5-HT2 receptor antagonist ritanserin (0.1 μM) or the 5-HT4 receptor antagonist SB 204070 (0.1 μM). However, the mucosally applied 5-HT3 receptor antagonists ondansetron (5 μM) and granisetron (1 μM) shifted the response curves to mucosally applied 5-HT to the right in a parallel and surmountable manner. The pD2 values in the absence and presence of ondansetron were 5.42 and 4.12, resp., and that of granisetron were 5.45 and 4.50 resp.,. Serosally applied ondansetron (5 μM) or granisetron (1 μM) had no effect on the concentration - response curve to mucosally applied 5-HT. However, the serosally applied ondansetron and granisetron antagonized the facilitatory effect of serosally applied 5-HT (10 μM) when administered in the presence of serosally applied SB 204070 (0.1 μM). It is concluded that the facilitatory effect of mucosally applied 5-HT to reduce the peristaltic threshold in the guinea pig ileum is mediated via a 5-HT3 receptor located on the mucosal and not the serosal side of the ileum.

OS.CITING REF COUNT: 20 THERE ARE 20 CAPLUS RECORDS THAT CITE THIS RECORD (20 CITINGS)
REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 26 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:674650 CAPLUS
DOCUMENT NUMBER: 127:341347
ORIGINAL REFERENCE NO.: 127:66843a,66846a
TITLE: Nonlinear intestinal absorption of 5-hydroxytryptamine receptor antagonist caused by absorptive and secretory transporters
AUTHOR(S): Tamai, Ikumi; Saheki, Ayaka; Saitoh, Ryoichi; Sai, Yoshimichi; Yamada, Ichimaro; Tsuji, Akira
CORPORATE SOURCE: Faculty of Pharmaceutical Sciences, Kanazawa University, Kanazawa, 920, Japan
SOURCE: Journal of Pharmacology and Experimental Therapeutics (1997), 283(1), 108-115
CODEN: JPETAB; ISSN: 0022-3565
PUBLISHER: Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The mechanism of the nonlinear concentration dependence of intestinal absorption of the 5-hydroxytryptamine receptor antagonist azasetron was studied by use of rat in situ intestinal perfusion, as well as an in vitro Ussing-type chamber method mounted with rat intestinal tissue and cultured monolayers of human adenocarcinoma Caco-2 cells. The intestinal absorption rate constant of azasetron evaluated by the Doluisio method increased significantly with increasing concentration of azasetron up to 10 mM in a nonlinear fashion and tended to decrease at higher concns. Mucosal-to-serosal directed permeation of [14C] azasetron across rat ileal sheets evaluated by the in vitro Ussing-type chamber method also increased in a nonlinear fashion in a low concentration range, followed by a decrease as the concentration was further increased, whereas serosal-to-mucosal directed permeation decreased in a concentration-dependent manner. Vectorial transport of [14C]azasetron across a Caco-2 cell monolayer was observed, with higher transport in the basolateral-to-apical direction at a trace concentration of azasetron. When the initial uptake rate of azasetron by Caco-2 cells was measured, it was saturable with an apparent half-saturation concentration of 15 mM and was reduced in the presence of several cationic compds. These observations suggest that azasetron is taken up by a carrier-mediated transport mechanism across the intestinal epithelial cells. When the steady-state uptake of [14C]azasetron was measured, it was increased in the presence of unlabeled azasetron and ondansetron. In addition, the steady-state uptake was enhanced in the presence of a P-glycoprotein inhibitor, cyclosporin A, and by ATP-depletion of the cells, although these treatments had no effect on the initial uptake of [14C]azasetron. Furthermore, the multidrug-resistant cancer cell line K562/ADM that overexpresses P-glycoprotein accumulated azasetron less extensively than did the parental drug-sensitive K562 cells. These results strongly suggest that azasetron is secreted into the intestinal lumen predominantly by P-glycoprotein. We conclude that intestinal transport of azasetron involves specialized transporters in both the absorptive and secretory directions, and the complex nonlinear intestinal absorption characteristics can be ascribed to the participation of multiple transport mechanisms.
OS.CITING REF COUNT: 32 THERE ARE 32 CAPLUS RECORDS THAT CITE THIS

RECORD (32 CITINGS)
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 27 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:568665 CAPLUS
DOCUMENT NUMBER: 127:215456
ORIGINAL REFERENCE NO.: 127:41788h,41789a
TITLE: 5-Hydroxytryptamine inhibits Na absorption and stimulates Cl secretion across canine tracheal epithelial sheets
AUTHOR(S): Tamaoki, J.; Chiyotani, A.; Takemura, H.; Konno, K.
CORPORATE SOURCE: First Department of Medicine, Tokyo Women's Medical College, Tokyo, 162, Japan
SOURCE: Clinical and Experimental Allergy (1997), 27(8), 972-977
CODEN: CLEAEN; ISSN: 0954-7894
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English
AB 5-Hydroxytryptamine (5-HT) can be released from mast cells and platelets through an IgE-dependent mechanism and may play a role in the pathogenesis of allergic bronchoconstriction. However, the effect of 5-HT on ion transport by airway epithelium remains uncertain. To determine whether 5-HT alters elec. and ion transport properties of Cl-secreting epithelia and, if so, what subtype of 5-HT receptors is involved, the authors studied canine tracheal epithelium under short-circuit conditions in vitro. Canine tracheal mucosa was mounted in Lucite half-chambers and the responses of short-circuit current (Is.c.), transepithelial PD and tissue conductance (G) were measured. In addition, ion fluxes were directly measured using 22Na and 36Cl. Mucosal addition of 5-HT caused a rapid increase in Is.c., which was accompanied by the increases in PD and G, whereas submucosal 5-HT had no effect. In the presence of amiloride, 5-HT and its receptor agonists dose-dependently increased Is.c., with the rank order of potency being 5-HT>α-methyl-5-HT>2-methyl-5HT>5-carboxamidotryptamine. The effect of 5-HT was inhibited by ketanserin and spiperone but not by ondansetron. 5-HT increased Cl flux from the submucosa to the mucosa with a slight inhibition of Na flux to the opposite direction. 5-HT inhibits airway epithelial Na absorption and stimulates Cl secretion. The latter action predominates the former and is mediated by 5-HT2 receptors. These effects may result in the increase in water movement toward the airway lumen.
OS.CITING REF COUNT: 9 THERE ARE 9 CAPLUS RECORDS THAT CITE THIS RECORD (9 CITINGS)
REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 28 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1997:557633 CAPLUS
DOCUMENT NUMBER: 127:239118
ORIGINAL REFERENCE NO.: 127:46553a,46556a
TITLE: Drug delivery systems containing ester sunscreens and penetration enhancers
INVENTOR(S): Reed, Barry Leonard; Morgan, Timothy Matthias; Finnin, Barrie Charles
PATENT ASSIGNEE(S): Monash University, Australia
SOURCE: PCT Int. Appl., 70 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9729735	A1	19970821	WO 1997-AU91	19970219	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU					
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
CA 2244089	A1	19970821	CA 1997-2244089	19970219	<--
CA 2244089	C	20090602			
AU 9717134	A	19970902	AU 1997-17134	19970219	<--
AU 706967	B2	19990701			
EP 901368	A1	19990317	EP 1997-904304	19970219	<--
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JP 2000504697	T	20000418	JP 1997-528834	19970219	<--
JP 4213211	B2	20090121			
AT 324865	T	20060615	AT 1997-904304	19970219	
EP 1674068	A1	20060628	EP 2005-22951	19970219	
EP 1674068	B1	20081008			
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ES 2262173	T3	20061116	ES 1997-904304	19970219	
EP 1769785	A1	20070404	EP 2006-25287	19970219	
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AT 410136	T	20081015	AT 2005-22951	19970219	
ES 2314538	T3	20090316	ES 2005-22951	19970219	
US 6299900	B1	20011009	US 1998-125436	19981218	<--
HK 1018884	A1	20060804	HK 1999-103965	19990911	
AU 9952589	A	19991202	AU 1999-52589	19991001	<--
US 20020028235	A1	20020307	US 2001-910780	20010724	<--
US 6818226	B2	20041116			
US 20040009214	A1	20040115	US 2003-428017	20030502	
US 6964777	B2	20051115			
US 20040013620	A1	20040122	US 2003-428016	20030502	
US 6929801	B2	20050816			
US 20040013621	A1	20040122	US 2003-428019	20030502	
US 6916487	B2	20050712			
US 20040028625	A1	20040212	US 2003-428012	20030502	
US 6916486	B2	20050712			
US 20040028725	A1	20040212	US 2003-428018	20030502	
US 6923983	B2	20050802			
US 20040096405	A1	20040520	US 2003-636976	20030808	
US 6998138	B2	20060214			
US 20040081684	A1	20040429	US 2003-644085	20030820	
US 7094422	B2	20060822			
US 20040146469	A1	20040729	US 2004-759303	20040120	
US 7438203	B2	20081021			
HK 1087355	A1	20090109	HK 2006-109387	20060824	
US 20070071803	A1	20070329	US 2006-513342	20060831	
US 20070077288	A1	20070405	US 2006-517575	20060908	
US 7387789	B2	20080617			
JP 2007326867	A	20071220	JP 2007-185782	20070717	
US 20080152597	A1	20080626	US 2007-905926	20071005	
US 20080131494	A1	20080605	US 2007-978556	20071030	

PRIORITY APPLN. INFO.: AU 1996-8144 A 19960219

	AU 1997-17134	A3 19970219
	EP 1997-904304	A3 19970219
	EP 2005-22951	A3 19970219
	JP 1997-528834	A3 19970219
	WO 1997-AU91	W 19970219
	US 1998-125436	A3 19981218
	US 2001-910780	A3 20010724
	US 2004-759303	A1 20040120

OTHER SOURCE(S): MARPAT 127:239118

AB A transdermal drug delivery system which comprises at least one physiol. active agent or prodrug thereof and at least one dermal penetration enhancer; characterized in that the dermal penetration enhancer is a safe skin-tolerant ester sunscreen. A non-occlusive, percutaneous or transdermal drug delivery system which comprises: (1) an effective amount of at least one physiol. active agent or prodrug thereof; (2) at least one non-volatile dermal penetration enhancer; and (3) at least one volatile liquid; characterized in that the dermal penetration enhancer is adapted to transport the physiol. active agent across a dermal surface or mucosal membrane of an animal, including a human, when the volatile liquid evaps., to form a reservoir or depot of a mixture comprising the penetration enhancer and the physiol. active agent or prodrug within said surface or membrane; and the dermal penetration enhancer is of low toxicity to, and is tolerated by, the dermal surface or mucosal membrane of the animal. The mean flux of 2% ketoprofen in 70% volume/volume aqueous ethanol through shed snakes kinetics in presence of 2% octyl salicylate in 70% volume/volume aqueous ethanol was 27.66 as compared to 2.58 μg/cm2.h for azone. A transdermal aerosol contained 17β-estradiol 2, octyl dimethyl-p-aminobenzoate 8, ethanol 69, and di-Me ether 30%.

OS.CITING REF COUNT: 30 THERE ARE 30 CAPLUS RECORDS THAT CITE THIS RECORD (32 CITINGS)

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 29 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1997:194051 CAPLUS

DOCUMENT NUMBER: 126:207379

ORIGINAL REFERENCE NO.: 126:39965a,39968a

TITLE: Gastric motility and mucosal ulcerogenic responses induced by prokinetic drugs in rats under prostaglandin-deficient conditions

AUTHOR(S): Takeuchi, Koji; Kato, Shinichi; Hirata, Takuya; Nishiwaki, Hidekazu

CORPORATE SOURCE: Department of Pharmacology & Experimental Therapeutics, Kyoto Pharmaceutical University, Kyoto, 607, Japan

SOURCE: Digestive Diseases and Sciences (1997), 42(2), 251-258
CODEN: DDSCDJ; ISSN: 0163-2116

PUBLISHER: Plenum

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Expts. were performed to examine whether gastric-prokinetic drugs may induce damage in the rat stomach under normal and prostaglandin (PG)-deficient conditions. Rats fasted for 18 h were s.c. administered 3 prokinetic drugs: metoclopramide (3-60 mg/kg), ondansetron (0.3-3 mg/kg), and cisapride (3-30 mg/kg). Half of these animals were pretreated with indomethacin (5 mg/kg) s.c. for induction of PG deficiency in the stomach. Administration of these drugs increased gastric motor activity in a dose-dependent manner and expedited gastric emptying at lower doses than those affecting gastric motility; the potency of the hypermotility effect was in the order: metoclopramide = ondansetron > cisapride. None of these drugs alone caused gross

damages in the stomach, although whitish rough areas were observed in the gastric mucosa along the folds. In the rats pretreated with indomethacin, however, both metoclopramide and ondansetron provoked multiple hemorrhagic lesions in the gastric mucosa. Given alone, indomethacin at this dose produced >90% inhibition of cyclooxygenase activity without causing any damage in the stomach; this PG-reducing effect was not affected by coadministration with the prokinetic drugs. The mucosal ulcerogenic responses induced by metoclopramide in the presence of indomethacin were inhibited by prior administration of atropine (1 mg/kg) or PGE2 (300 μg/kg), at doses that inhibited the gastric hypermotility induced by metoclopramide. These results suggest that: (1) gastric-prokinetic drugs induce damage in rat stomachs under PG-deficient conditions at doses that enhance gastric motility and emptying but not at doses that expedite gastric emptying only; (2) gastric hypermotility has the potential to cause gross damage in the stomach, supporting the importance of gastric motility as a pathogenic element of gastric lesions.

OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)

L4 ANSWER 30 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1995:942183 CAPLUS
DOCUMENT NUMBER: 123:330657
ORIGINAL REFERENCE NO.: 123:59081a,59084a
TITLE: Serotonin causes acute gastric mucosal injury in rats, probably via 5HT1D receptors
AUTHOR(S): Gidener, Sedef; Apaydin, Sebnem; Kupelioglu, Ali; Guven, Hulya; Gelal, Ayse; Gure, Ataman
CORPORATE SOURCE: Medical Faculty, Dokuz Eylul University, Izmir, 35340, Turk.
SOURCE: International Journal of Experimental Pathology (1995), 76(4), 237-40
CODEN: IJEPEI; ISSN: 0959-9673
PUBLISHER: Blackwell
DOCUMENT TYPE: Journal
LANGUAGE: English

AB 5-HT-induced acute gastric mucosal injury was assessed in rats by using 5HT1, 5HT2, 5HT3, 5HT4 or muscarinic receptor related drugs. Rats were treated with antagonists i.p. and 30 min later either vehicle, 5-HT (20 mg/kg) or other agonists were administered s.c. The stomachs were removed 4 h after the last injection and mucosal integrity was assessed by light microscopy using a histol. ulcer index (HUI). The HUI was significantly increased following 5-HT administration (1.57) when compared with controls (0.14). 5HT1 agonist 5-carboxamidotryptamine (20 mg/kg) produced acute gastric erosion and increased the HUI. The HUI in the animals receiving 5-HT1D agonist sumatriptan (7 mg/kg) was 1.62. 5HT2 antagonist ketanserin (2.5-15 mg/kg), 5HT3 antagonist ondansetron (1-5 mg/kg), 5HT4 antagonist DAU 6285 (1-10 mg/kg) and atropine (1.5-30 mg/kg) exerted no effect whereas 5HT1/2 antagonist metitepine (0.05-0.5 mg/kg) caused a dose dependent inhibition of the effect of 5-HT. The results from this study demonstrate that 5-HT causes acute gastric mucosal injury and this injury is probably due to the activation of the 5-HT1D receptors.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L4 ANSWER 31 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1995:805092 CAPLUS
DOCUMENT NUMBER: 123:246511
ORIGINAL REFERENCE NO.: 123:43763a,43766a
TITLE: The influence of peripheral or central administration of ondansetron on stress-induced gastric

ulceration in rats
AUTHOR(S): Ogle, C. W.; Hui, S.-C. G.
CORPORATE SOURCE: Fac. Med., Univ. Hong Kong, Hong Kong
SOURCE: Experientia (1995), 51(8), 786-9
CODEN: EXPEAM; ISSN: 0014-4754
PUBLISHER: Birkhaeuser
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ondansetron (0.08, 0.15 or 0.3 mg/kg) injected s.c., every 12 h with the fourth dose given 0.5 h before expts., dose-dependently lessened gastric glandular mucosal ulceration produced by cold-restraint stress for 2 h. When given intracerebrally (i.c) (0.1, 0.5 or 1 μg), using the same treatment regimen, infusion of ondansetron 1 μg into the nucleus amylgdaloideus centralis decreased stress-evoked ulcers; in contrast, injection of the same dose into the nucleus accumbens intensified these lesions. The associated stress-induced stomach wall mast cells degranulation was unaffected by all s.c. or i.c. doses of ondansetron. Pretreatment with disodium cromoglycate i.p. alone, or concurrently with ondansetron s.c., prevents not only ulceration but also mast cell degranulation. 5-Hydroxytryptamine3 receptor antagonism appears to inhibit stress-evoked ulcers mainly by blocking the peripheral effects of amine after its release from the gastric mucosal mast cells.
OS.CITING REF COUNT: 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

L4 ANSWER 32 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:549635 CAPLUS
DOCUMENT NUMBER: 121:149635
ORIGINAL REFERENCE NO.: 121:26853a,26856a
TITLE: Modulatory role of 5-HT3 receptors in gastric function and ethanol-induced mucosal damage in rat stomachs
AUTHOR(S): Cho, C. H.; Koo, M. W. L.; Ko, J. K. S.
CORPORATE SOURCE: Fac. Med., Univ. Hong Kong, Hong Kong
SOURCE: Pharmacology (1994), 49(3), 137-43
CODEN: PHMGBN; ISSN: 0031-7012
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The involvement of 5-hydroxytryptamine (5-HT) in gastric function and mucosal damage has been defined. 5-HT also potentiates lesion formation in animals. The current study investigated further whether these actions are mediated through 5-HT3 receptors in rats. Ondansetron, a 5-HT3 receptor antagonist, was given s.c., 2 or 4 mg/kg, 30 min before the gastric parameters were measured. The higher dose of ondansetron increased gastric mucosal blood flow (GMBF) and also basal acid and Na+ secretion. However, it id not affect pepsin output. 5-HT time dependently reduced GMBF and pepsin secretion, but not that of acid and Na+. These actions were not altered by ondansetron pretreatment. The drug, however, dose dependently reduced ethanol-induced gastric mucosal lesions in the 5-HT-treated animals. These findings indicate that 5-HT3 receptors regulate not only basal GMBF, but also acid and Na+ secretion in stomachs. However, the depressive action of 5-HT on GMBF and pepsin secretion is most likely not mediated through 5-HT3 receptors. Ondansetron also modulates the toxicities of ethanol in the stomach and this action is likely to be mediated through the preservation of GMBF.
OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

L4 ANSWER 33 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:400551 CAPLUS

DOCUMENT NUMBER: 121:551
ORIGINAL REFERENCE NO.: 121:119a,122a
TITLE: 5-Hydroxytryptamine3-receptor blockade protects against gastric mucosal damage in rats
AUTHOR(S): Ogle, C.W.; Hui, S-C.G.; Qiu, B.S.; Li, K.M.
CORPORATE SOURCE: Fac. Med., Univ. HONG KONG, HONG KONG, Hong Kong
SOURCE: Acta Physiologica Hungarica (1992), 80(1-4), 181-8
CODEN: APHHDU; ISSN: 0231-424X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ondansetron, a specific 5-hydroxytryptamine3 (5-HT3)-blocker, injected s.c. (0.038, 0.075, 0.15 or 0.3 mg/kg) every 12 h with the fourth dose given 0.5 h before restraint at 4°C (stress) or oral administration (p.o.) of 1 mL 80% ethanol, dose-dependently prevented gastric mucosal damage in female Sprague-Dawley rats (160-180 g); the animals were killed 2 or 1 h after stress or ethanol p.o., resp. A similar pretreatment regimen with cyproheptadine (0.1, 0.25 or 0.5 mg/kg) or ketanserin (15, 30, or 75 μg/kg), both being 5HT2-receptor antagonists, also dose-dependently lowered the severity of stress- or ethanol-induced mucosal lesions. Only the higher doses of phenobarbitone (25 or 50 mg/kg given s.c. in a single dose 0.5 h beforehand) inhibited stress-induced gastric ulcers; however, even the lowest non-antiulcer dose (12.5 mg/kg), effectively produced CNS depression. These preliminary findings suggest that 5HT3-receptor blockade not only can antagonize stress- or ethanol-evoked gastric mucosal damage, but also may act through a peripheral mechanism.
OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L4 ANSWER 34 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:235356 CAPLUS
DOCUMENT NUMBER: 120:235356
ORIGINAL REFERENCE NO.: 120:41393a,41396a
TITLE: Use of Caco-2 cells as an in vitro intestinal absorption and metabolism model
AUTHOR(S): Gan, Liang Shang; Eads, Cindy; Niederer, Tara; Bridgers, Avis; Yanni, Souzan; Hsyu, Poe Hirr; Pritchard, Fred J.; Thakker, Dhiren
CORPORATE SOURCE: Dep. Drug Metabolism, Glaxo Inc. Res. Inst., Research Triangle Park, NC, 27709, USA
SOURCE: Drug Development and Industrial Pharmacy (1994), 20(4), 615-31
CODEN: DDIPD8; ISSN: 0363-9045
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The Caco-2 cell line, a human colorectal carcinoma cell line, is an established in vitro model for the study of drug transport in the human intestine. The authors have routinely utilized this in vitro model to 1) elucidate intestinal absorption mechanisms of small drug mols. and peptide-like therapeutic agents (e.g. paracellular/transcellular passive diffusion and carrier-mediated active transport), 2) screen and select orally active therapeutic agents, 3) identify optimum luminal pH's for drug absorptions, 4) address dissoln. rate-related absorption problems, 5) assess mucosal toxicity of therapeutic agents, and 6) evaluate prodrug approaches for enhanced drug absorptions. The authors have also utilized this in vitro model to assess the metabolic stability of therapeutic agents in the intestinal epithelium. demonstrated in this report are primarily the techniques for the elucidation of absorption mechanisms. Examples of the characterization of paracellular/transcellular passive diffusion pathways and carrier-mediated active transport will be given. Application of the Caco-2 model to the

process of drug development will also be discussed.
OS.CITING REF COUNT: 27 THERE ARE 27 CAPLUS RECORDS THAT CITE THIS RECORD (27 CITINGS)

L4 ANSWER 35 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 1994:226984 CAPLUS
DOCUMENT NUMBER: 120:226984
ORIGINAL REFERENCE NO.: 120:40121a,40124a
TITLE: Compositions of oral nondissolvable matrixes for transmucosal administration of medicaments
INVENTOR(S): Stanley, Theodore H.; Hague, Brian
PATENT ASSIGNEE(S): University of Utah Research Foundation, USA
SOURCE: U.S., 20 pp. Cont.-in-part of U.S. 4,863,737.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 9
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5288498	A	19940222	US 1989-403752	19890905 <--
US 4671953	A	19870609	US 1985-729301	19850501 <--
EP 487520	A1	19920603	EP 1989-909497	19890816 <--
EP 487520	B1	19950412		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
JP 05501539	T	19930325	JP 1989-504878	19890816 <--
JP 2801050	B2	19980921		
AU 641127	B2	19930916	AU 1989-40704	19890816 <--
AT 120953	T	19950415	AT 1989-909497	19890816 <--
CA 1338978	C	19970311	CA 1989-609378	19890824 <--
AU 9050352	A	19910408	AU 1990-50352	19890905 <--
AU 645966	B2	19940203		
EP 493380	A1	19920708	EP 1990-902584	19890905 <--
EP 493380	B1	19971029		
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE				
US 5132114	A	19920721	US 1989-402881	19890905 <--
JP 05501854	T	19930408	JP 1990-502779	19890905 <--
CA 1339075	C	19970729	CA 1989-610329	19890905 <--
AT 159658	T	19971115	AT 1990-902584	19890905 <--
CA 2066403	A1	19910306	CA 1990-2066403	19900803 <--
CA 2066403	C	19980414		
WO 9103236	A1	19910321	WO 1990-US4369	19900803 <--
W: AU, CA, JP, NO				
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE				
AU 9063371	A	19910408	AU 1990-63371	19900803 <--
AU 642664	B2	19931028		
EP 490944	A1	19920624	EP 1990-913359	19900803 <--
EP 490944	B1	19960529		
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE				
JP 05500058	T	19930114	JP 1990-512483	19900803 <--
JP 2749198	B2	19980513		
AT 138562	T	19960615	AT 1990-913359	19900803 <--
ES 2089027	T3	19961001	ES 1990-913359	19900803 <--
NO 9200565	A	19920213	NO 1992-565	19920213 <--
NO 304056	B1	19981019		
DK 9200193	A	19920214	DK 1992-193	19920214 <--
DK 175779	B1	20050214		
NO 9200858	A	19920304	NO 1992-858	19920304 <--
NO 9200855	A	19920410	NO 1992-855	19920304 <--
NO 9200854	A	19920427	NO 1992-854	19920304 <--
DK 9200300	A	19920505	DK 1992-300	19920305 <--

DK 175773	B1	20050214			
AU 9460697	A	19940623	AU 1994-60697	19940427	<--
US 5855908	A	19990105	US 1994-339655	19941115	<--
PRIORITY APPLN. INFO.:			US 1985-729301	A2 19850501	
			US 1987-60045	A2 19870608	
			EP 1989-909497	A 19890816	
			WO 1989-US3518	W 19890816	
			US 1989-403752	A 19890905	
			WO 1989-US3801	A 19890905	
			WO 1990-US4369	A 19900803	
			US 1993-152414	B1 19931112	

AB Compns. and methods of manufacture for producting a medicament composition capable of absorption through the mucosal tissues of the mouth, pharynx, and esophagus are disclosed. The present invention relates to such compns. and methods which are useful in administering lipophilic and nonlipophilic drugs in a dose-to-effect manner such that sufficient drug is administered to produce precisely a desired effect. The invention also relates to manufacturing techniques that enable therapeutic agents to be incorporated into nondissolvable drug containment matrixes which are capable of releasing the drug within a patient's mouth. An appliance or holder is preferably attached to the drug containment matrix. Employing the present invention the drug may be introduced into the patient's bloodstream almost as fast as through injection, and much faster than using the oral administration route, while avoiding the neg. aspects of both of these methods. The nondissolvable drug containment matrix may include permeation enhancers to increase the drug adsorption by the mucosal tissues of the mouth. The matrix composition may also include pH buffering agents to modify the saliva pH thereby increasing the absorption of the drug through the mucosal tissues. Figures show views of some dosage forms.

OS.CITING REF COUNT: 22 THERE ARE 22 CAPLUS RECORDS THAT CITE THIS RECORD (26 CITINGS)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 36 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1994:226981 CAPLUS
DOCUMENT NUMBER: 120:226981
ORIGINAL REFERENCE NO.: 120:40120h,40121a
TITLE: Compositions of oral dissolvable medicaments
INVENTOR(S): Stanley, Theodore H.; Hague, Brian
PATENT ASSIGNEE(S): University of Utah, USA
SOURCE: U.S., 22 pp. Cont.-in-part of U.S. 4,863,737.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 9
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 5288497	A	19940222	US 1989-403751	19890905	<--
US 4671953	A	19870609	US 1985-729301	19850501	<--
EP 487520	A1	19920603	EP 1989-909497	19890816	<--
EP 487520	B1	19950412			
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE					
JP 05501539	T	19930325	JP 1989-504878	19890816	<--
JP 2801050	B2	19980921			
AU 641127	B2	19930916	AU 1989-40704	19890816	<--
AT 120953	T	19950415	AT 1989-909497	19890816	<--
CA 1338978	C	19970311	CA 1989-609378	19890824	<--

AU 9050352	A	19910408	AU 1990-50352	19890905	<--
AU 645966	B2	19940203			
EP 493380	A1	19920708	EP 1990-902584	19890905	<--
EP 493380	B1	19971029			
R: AT, BE, CH, DE, FR, GB, IT, LI, LU, NL, SE					
US 5132114	A	19920721	US 1989-402881	19890905	<--
JP 05501854	T	19930408	JP 1990-502779	19890905	<--
CA 1339075	C	19970729	CA 1989-610329	19890905	<--
AT 159658	T	19971115	AT 1990-902584	19890905	<--
CA 2066423	A1	19910306	CA 1990-2066423	19900803	<--
CA 2066423	C	19980414			
WO 9103237	A1	19910321	WO 1990-US4384	19900803	<--
W: AU, CA, JP, NO					
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE					
AU 9062877	A	19910408	AU 1990-62877	19900803	<--
AU 645265	B2	19940113			
EP 490916	A1	19920624	EP 1990-912733	19900803	<--
EP 490916	B1	19951018			
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE					
JP 05503917	T	19930624	JP 1990-512229	19900803	<--
EP 630647	A1	19941228	EP 1994-111352	19900803	<--
EP 630647	B1	19990303			
R: AT, BE, CH, DE, DK, ES, FR, GB, IT, LI, LU, NL, SE					
AT 129148	T	19951115	AT 1990-912733	19900803	<--
ES 2077686	T3	19951201	ES 1990-912733	19900803	<--
AT 177007	T	19990315	AT 1994-111352	19900803	<--
ES 2133448	T3	19990916	ES 1994-111352	19900803	<--
NO 9200565	A	19920213	NO 1992-565	19920213	<--
NO 304056	B1	19981019			
DK 9200193	A	19920214	DK 1992-193	19920214	<--
DK 175779	B1	20050214			
NO 9200857	A	19920406	NO 1992-857	19920304	<--
NO 304348	B1	19981207			
NO 9200855	A	19920410	NO 1992-855	19920304	<--
NO 9200854	A	19920427	NO 1992-854	19920304	<--
DK 9200300	A	19920505	DK 1992-300	19920305	<--
DK 175773	B1	20050214			
AU 9455218	A	19940428	AU 1994-55218	19940218	<--
AU 668004	B2	19960418			
AU 9460697	A	19940623	AU 1994-60697	19940427	<--
US 5824334	A	19981020	US 1996-636828	19960419	<--
US 5783207	A	19980721	US 1997-795359	19970204	<--
US 5785989	A	19980728	US 1997-822560	19970319	<--

PRIORITY APPLN. INFO.:

US 1985-729301	A2	19850501
US 1987-60045	A2	19870608
EP 1989-909497	A	19890816
WO 1989-US3518	W	19890816
US 1989-403751	A	19890905
WO 1989-US3801	A	19890905
EP 1990-912733	A3	19900803
WO 1990-US4384	A	19900803
US 1993-152396	B1	19931112
US 1994-333233	B2	19941102
US 1995-439127	B1	19950511

AB Compns. and methods of manufacture for producing a medicament composition capable of absorption through the mucosal tissues of the mouth, pharynx, and esophagus are disclosed. The present invention relates to such compns. and methods which are useful in administering lipophilic and nonlipophilic drugs in a dose-to-effect manner that sufficient drug is administered to produce precisely a desired effect. The invention also relates to a manufacturing technique that enables a therapeutic agent or drug to

be incorporated into a flavored dissolvable matrix. An appliance or holder is preferably attached to the dissolvable matrix. Employing the present invention the drug may be introduced into the patient's bloodstream almost as fast as through injection, and much faster than using the oral administration route, while avoiding the neg. aspects of both of these methods. The present invention achieves these advantages by incorporating the drug into a carbohydrate, fat, protein, wax, or other dissolvable matrix composition The dissolvable matrix may include permeation enhancers to increase the drug absorption by the mucosal tissues of the mouth. The matrix composition may also include pH buffering agents to modify the salival pH thereby increasing the absorption of the drug through the mucosal tissue. Methohexital sodium was incorporated into a dissolvable matrix including citric acid; ribotide; Compritol 888; aspartame; vanilla, wild cherry, and peppermint microcapsules; compressible sugar; and maltodextrin.

OS.CITING REF COUNT: 40 THERE ARE 40 CAPLUS RECORDS THAT CITE THIS RECORD (45 CITINGS)

REFERENCE COUNT: 75 THERE ARE 75 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 37 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1994:420 CAPLUS

DOCUMENT NUMBER: 120:420

ORIGINAL REFERENCE NO.: 120:99a,102a

TITLE: RS 23597-190: a potent and selective 5-HT4 receptor antagonist

AUTHOR(S): Eglen, R. M.; Bley, K.; Bonhaus, D. W.; Clark, R. D.; Hegde, S. S.; Johnson, L. G.; Leung, E.; Wong, E. H. F.

CORPORATE SOURCE: Inst. Pharmacol., Syntex Discovery Res., Palo Alto, CA, 94304, USA

SOURCE: British Journal of Pharmacology (1993), 110(1), 119-26
CODEN: BJPCBM; ISSN: 0007-1188

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The pharmacol. properties of RS 23597-190 (3-(piperidin-1-yl)propyl-4-amino-5-chloro-2-methoxybenzoate hydrochloride) have been studied in vitro and in vivo. RS 23597-190 competitively antagonized 5-HT4 receptor-mediated relaxations of rat, carbachol precontracted esophageal muscularis mucosae, (pA2 = 7.8; Schild slope = 1.2). Affinity ests. (-log KB) at 5-HT4 receptors using either renzapride or SC-53116 as agonists yielded a -log KB value of 8.0. In contrast, RS 23597-190 failed to antagonize contractile responses to 5-HT of guinea-pig ileal 5-HT3 receptors, even at concns. up to 10 μM. Increases in short-circuit current, induced by 5-HT, were studied in guinea-pig ileal mucosal sheets. Concentration-response curves to 5-HT were biphasic, with the high potency phase to 5-HT inhibited by RS 23597-190 and mimicked by 5-methoxytryptamine. The -log KB value for RS 23597-190 at the high potency phase was 7.3 confirming that 5-HT4 receptors mediated the high potency phase. In rat isolated vagus nerve, 5-HT elicited a slow, maintained depolarization at low concns. and a rapid, transient depolarization at higher concns. The high potency, slow depolarizing phase to 5-HT was abolished selectively in the presence of 1 μM RS 23597-190 and the low potency phase was abolished selectively in the presence of 1 μM ondansetron. These data confirm that 5-HT4 and 5-HT3 receptors mediated slow and fast depolarization responses, resp. At 5-HT3 binding sites in membranes from NG 108-15 cells, labeled by [3H]-quipazine, RS 23597-190 exhibited an apparent affinity (-log Ki) of 5.7. At 5-HT3 receptors in membranes from rat cerebral cortex, labeled by [3H]-RS 42358-197, the apparent affinity (-log Ki) of RS 23597-190 was also 5.7. In both studies, Hill coeffs. were not significantly different

from unity. At 5-HT1A, 5-HT2, muscarinic M1, M2, M3, M4 and dopamine D1 and D2 receptors, RS 23597-190 exhibited low apparent affinities, with all -log Ki values less than 5.5. I.v. infusion of RS 23597-190 in the conscious, restrained rat antagonized the von Bezold Jarisch reflex induced by 2-Me-5-HT, with an ID50 of 300 μg kg-1 min-1, i.v. In the anesthetized, bilaterally vagotomized micropig, RS 23597-190 (6 mg kg-1, i.v.) antagonized 5-HT-induced tachycardia with a half-life of 77 (63-99) min. Transient arrhythmic effects were noted after administration of the compound In conclusion, RS 23597-190 acts as a high affinity, selective competitive antagonist at 5-HT4 receptors. Thus, the compound appears to be a useful tool for 5-HT4 receptor identification in vitro. In vivo, the compound is rapidly metabolized in pigs such that 5-HT4 blockade is not maintained. However, in the rat, when given by infusion, RS 23597-190 antagonizes 5-HT3 mediated responses, at doses consistent with a low affinity 5-HT3 receptor. These data suggest that, under appropriate exptl. conditions, RS 23597-190 may also be used in vivo to characterize further 5-HT4 receptor function.

OS.CITING REF COUNT: 20 THERE ARE 20 CAPLUS RECORDS THAT CITE THIS RECORD (20 CITINGS)

L4 ANSWER 38 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1993:663069 CAPLUS

DOCUMENT NUMBER: 119:263069

ORIGINAL REFERENCE NO.: 119:46825a,46828a

TITLE: Short-circuit current responses to 5-hydroxytryptamine in human ileal mucosa are mediated by a 5-HT4 receptor

AUTHOR(S): Burleigh, David E.; Borman, Richard A.

CORPORATE SOURCE: Dep. Pharmacol., Queen Mary Westfield Coll., London, E1 4NS, UK

SOURCE: European Journal of Pharmacology (1993), 241(1), 125-8
CODEN: EJPHAZ; ISSN: 0014-2999

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 5-Hydroxytryptamine (5-HT) increases short-circuit current when added to the serosal side of human isolated ileal mucosa; mucosally applied 5-HT was ineffective. Tetrodotoxin reduced both basal short-circuit current and increases in short-circuit current due to elec. field stimulation of mucosal nerves. However, neither tetrodotoxin, ondansetron nor methysergide plus ketanserin affected 5-HT-induced increases in short-circuit current. Application of SDZ 205-557 (2-diethylaminoethyl-(2-methoxy-4-amino-5-chloro)benzoate) to the tissue caused a significant increase in the concentration ratio between two successive 5-HT response curves. It is concluded that the effect of 5-HT on short-circuit current of human ileal mucosa appears to be due to stimulation of a 5-HT4 receptor.

OS.CITING REF COUNT: 24 THERE ARE 24 CAPLUS RECORDS THAT CITE THIS RECORD (24 CITINGS)

L4 ANSWER 39 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1993:16716 CAPLUS

DOCUMENT NUMBER: 118:16716

ORIGINAL REFERENCE NO.: 118:3029a,3032a

TITLE: Effects of 5-hydroxytryptamine and 5-hydroxytryptamine receptor agonists on ion transport across mammalian airway epithelia

AUTHOR(S): Graham, A.; Alton, E. W. F. W.; Geddes, D. M.

CORPORATE SOURCE: Ion Transp. Lab., Natl. Heart Lung Inst., London, SW3 6LR, UK

SOURCE: Clinical Science (1992), 83(3), 331-6
CODEN: CSCIAE; ISSN: 0143-5221

DOCUMENT TYPE: Journal

LANGUAGE: English

AB 5-HT and related compds. were studied to investigate whether any might be a useful alternative to amiloride for clin. use, and to further assess the possible physiol. role of 5-HT in the regulation of airway ion transport. Sheep tracheal epithelium was mounted in Ussing chambers under short-circuit conditions. Mucosal application of 5-HT resulted in an immediate, reversible, concentration-related decrease in the short-circuit current, maximal with 38% inhibition of the short-circuit current at 25 mM. This response was completely inhibited by pretreatment of tissues with mucosal amiloride (100 μM). These features are consistent with a direct effect of 5-HT on amiloride-sensitive sodium channels. Similar results were obtained in a limited number of studies using human bronchial epithelium. The 5-HT3 agonist 2-methyl-5-HT had no effect on the short-circuit current at concns. of up to 5 mM. The 5-HT1D agonist sumatriptan had no effect at concns. below 5 mM and at 5 mM had only a transient effect. The 5-HT1A agonists buspirone and 8-hydroxy-2-(di-n-propylamino)tetralin and the 5-HT2 agonist α-methyl-5-HT were all more potent inhibitors of the short-circuit current than 5-HT, but, although their effects were reduced by pretreatment of tissues with mucosal amiloride (100 μM), none had a specific effect on the amiloride-sensitive sodium current. The effect of buspirone on the short-circuit current was also studied after mucosal sodium substitution, and although its effect was again reduced, significant inhibition of the short-circuit current still occurred, indicating that ion transport processes other than sodium absorption were being affected. Mucosal application of ondansetron, an antagonist at the 5-HT3 receptor (an ion channel), also produced a dose-related inhibition of the short-circuit current that was not mediated via the amiloride-sensitive sodium current. Pretreatment of tissues with ondansetron had no effect on the subsequent response to 5-HT. Thus, mucosally applied 5-HT specifically inhibits amiloride-sensitive sodium transport in airway epithelia, but with a median inhibitory concentration too high for it to be therapeutically useful. The high median inhibitory concentration also indicates that 5-HT is unlikely to be a physiol. regulator of sodium channels. Screening a number of 5-HT receptor agonists has failed to identify a more potent inhibitor of sodium transport which may have had therapeutic potential.

OS.CITING REF COUNT: 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

L4 ANSWER 40 OF 52 CAPLUS COPYRIGHT 2009 ACS on STN

ACCESSION NUMBER: 1992:208452 CAPLUS

DOCUMENT NUMBER: 116:208452

ORIGINAL REFERENCE NO.: 116:35155a,35158a

TITLE: Role of the serotonin3 receptor in stress-induced defecation

AUTHOR(S): Miyata, Keiji; Kamato, Takeshi; Nishida, Akito; Ito, Hiroyuki; Yuki, Hidenobu; Yamano, Mayumi; Tsutsumi, Rie; Katsuyama, Yoshinori; Honda, Kazuo

CORPORATE SOURCE: Med. Res. Lab. I, Yamanouchi Pharm. Co. Ltd., Tsukuba, 305, Japan

SOURCE: Journal of Pharmacology and Experimental Therapeutics (1992), 261(1), 297-303
CODEN: JPETAB; ISSN: 0022-3565

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The possibility that 5-HT mediates bowel dysfunction caused by stress was evaluated in rats and mice treated with 5-HT or TRH injection and in rats subjected to stress. Restraint stress at room temperature (23°) increased fecal pellet output without the formation of gastrointestinal

mucosal lesions in free-feeding rats, and caused diarrhea in 90-100% of animals within 3 h in food-deprived rats. Oral YM060, ondansetron, granisetron, atropine, and diazepam and s.c. tetrodotoxin inhibited these stress-induced changes in bowel function in fed and fasted rats. Methysergide (s.c.) inhibited stress-induced diarrhea, and it had a partial effect on stress-induced increases in fecal pellet output. Exogenous 5-HT increased fecal pellet output in rats and caused diarrhea in mice. YM060, granisetron, atropine, and tetrodotoxin, but not methysergide, dose-dependently inhibited 5-HT-induced increases in fecal pellet output and 5-HT-induced diarrhea. S.c. TRH, an endogenous candidate in centrally mediated stress-induced bowel function responses, increased fecal pellet output. The change in bowel function induced by TRH was also reduced by oral YM060, granisetron, and atropine and by s.c. tetrodotoxin. In contrast, s.c. methysergide did not affect TRH-induced defecation. Thus, exogenous and endogenous 5-HT, whose release may be induced by TRH, appear to cause an increase in the number of stools excreted or diarrhea in rats or mice via the 5-HT3 receptor. Therefore, endogenous 5-HT may be one of the substances that mediate stress-induced responses of gastrointestinal function.

OS.CITING REF COUNT: 48 THERE ARE 48 CAPLUS RECORDS THAT CITE THIS RECORD (48 CITINGS)

L4 ANSWER 41 OF 52 MEDLINE on STN

ACCESSION NUMBER: 2004181293 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15075453
TITLE: Neural control of the release and action of secretin.
AUTHOR: Chey W Y; Chang T-M
CORPORATE SOURCE: Rochester Institute for Digestive Diseases and Sciences, Rochester, NY 14607, USA.. williamchey@ridds.org
SOURCE: Journal of physiology and pharmacology : an official journal of the Polish Physiological Society, (2003 Dec) Vol. 54 Suppl 4, pp. 105-12. Ref: 18
Journal code: 9114501. E-ISSN: 1899-1505.
PUB. COUNTRY: Poland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200801
ENTRY DATE: Entered STN: 13 Apr 2004
Last Updated on STN: 19 Dec 2004
Entered Medline: 17 Jan 2008

AB The release and physiological actions of secretin on pancreatic exocrine secretion and gastric secretion of acid and motility are regulated by neuro-hormonal control. The release of secretin by duodenal acidification is mediated by a secretin releasing peptide (SRP). The release and action of SRP are neurally mediated depending on vagal afferent pathway. SRP activity in acid perfusate of the duodenum was substantially decreased when rats were treated with tetradotoxin (TTX), perivagal application of capsaicin, a beta-adrenergic blocker, Met-enkephalin (MEK) or vagotomy. The release of secretin by SRP was abolished in rats treated with TTX, mucosal or perivagal application of capsaicin, MEK or vagotomy. Both release of secretin and pancreatic exocrine secretion (PES) elicited by duodenal acidification were also inhibited dose-dependently by Met-enkepahlin, 5-HT(2) antagonist, ketanserin and 5-HT(3) antagonist, ondansetron. Stimulation of PES and inhibition of gastric acid secretion and motility by secretin in a physiological dose are also dependent on the vagal afferent pathway as these effects of secretin are abolished by perivagal capsaicin treatment or vagotomy. In conscious rats, vagotomy, vagal ligation, or perivagal colchicine but not capsaicin treatment reduced the number of secretin binding sites in the forestomach

suggesting another mode of neural regulation that affects gastric motility. Except in the rat, stimulation of PES by secretin in a physiological dose is profoundly inhibited by atropine indicating the importance of a cholinergic input. In isolated and perfused rat pancreas, electrical field stimulation potentiated secretin-stimulated PES that was suppressed by atropine and anti-GRP serum, suggesting the roles of intrapancreatic cholinergic and GRP-containing neurons. In rats, secretin-stimulated PES was inhibited by a NO synthase inhibitor suggesting mediation by NO. However, the neuropeptides and neurotransmitters involved in regulation of the release and action of secretin and their sites of action remain to be elucidated.

L4 ANSWER 42 OF 52 MEDLINE on STN
ACCESSION NUMBER: 1995149908 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7847260
TITLE: A phase II trial of zeniplatin in metastatic melanoma.
AUTHOR: Olver I; Green M; Peters W; Zimet A; Toner G; Bishop J; Ketelbey W; Rastogi R; Birkhofer M
CORPORATE SOURCE: Peter MacCallum Cancer Institute, Melbourne, Victoria, Australia.
SOURCE: American journal of clinical oncology, (1995 Feb) Vol. 18, No. 1, pp. 56-8.
Journal code: 8207754. ISSN: 0277-3732.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CLINICAL TRIAL, PHASE II)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199503
ENTRY DATE: Entered STN: 16 Mar 1995
Last Updated on STN: 16 Mar 1995
Entered Medline: 7 Mar 1995
AB A third-generation platinum analogue, zeniplatin, was administered at a dose of 145 mg/m2 intravenously over 60-90 minutes every 21 days as the initial chemotherapy to 21 patients with metastatic melanoma. Prehydration and mannitol diuresis was introduced after the first 7 patients. There were 17 males and 4 females. The median age was 52 (range: 29-81). ECOG performance status was 0 in 10 patients, 1 in 8 patients and 2 in 3 patients. Major disease sites were lymph nodes, skin, lung, liver, and bone. Patients received a median of 2 cycles (range: 1-7). Two patients achieved partial responses. One with nodal disease progressed after 166 days and the other with buccal mucosal disease after 142 days. A third patient showed partial regression of nodal disease but developed cerebral metastases. Gastrointestinal toxicity included WHO grade 3 vomiting in 8 patients and nausea in 2. Antiemetics were used, but ondansetron was not available. WHO grade 3 hematologic toxicities included neutropenia in 8 patients and anemia and thrombocytopenia in 1 patient. Thrombocytosis was seen in 35% of courses. Dosage reduction was required in 15% of courses and escalation in 5% of courses. Three patients developed phlebitis related to the infusion. One patient developed a reversible rise in serum creatinine, but, unlike other studies, no severe nephrotoxicity was reported. Zeniplatin demonstrated only modest activity in melanoma with significant gastrointestinal and hematologic toxicity.

L4 ANSWER 43 OF 52 MEDLINE on STN
ACCESSION NUMBER: 1994323910 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8048005
TITLE: The 5-HT4 receptor mediates 5-hydroxytryptamine-induced rise in short circuit current in the human jejunum in

vitro.
AUTHOR: Budhoo M R; Kellum J M
CORPORATE SOURCE: Department of Surgery, Medical College Virginia, Richmond 23298.
CONTRACT NUMBER: DK 43899 (United States NIDDK NIH HHS)
SOURCE: Surgery, (1994 Aug) Vol. 116, No. 2, pp. 396-400.
Journal code: 0417347. ISSN: 0039-6060.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 9 Sep 1994
Last Updated on STN: 9 Sep 1994
Entered Medline: 30 Aug 1994

AB BACKGROUND. 5-Hydroxytryptamine (5-HT) is a potent intestinal secretagogue for chloride and a mediator of diarrhea in the carcinoid syndrome. 5-HT-induced chloride secretion is seen as a change in short circuit current (Isc) in muscle-stripped, chambered human jejunum. The aim of this study was to determine which 5-HT receptors mediate a 5-HT-induced change in Isc in the human jejunum. METHODS. Segments of jejunum obtained from patients (n = 23) having obesity surgery were stripped of muscularis, and the mucosal sheets were mounted in flux chambers and short-circuited. By a cumulative method, a 5-HT-induced change in Isc was measured in the presence or absence of 0.2 mumol/L of neural conduction inhibitor tetrodotoxin or 5-HT receptor antagonists (n = 4 to 5): 10 mumol/L 5-HTP-DP, a 5-HT1p antagonist; 0.1 mumol/L ketanserin, a 5-HT2 antagonist; 0.3 mumol/L ondansetron, a 5-HT3 antagonist; 0.05 and 1 mumol/L ICS 205-930, a selective 5-HT3 antagonist at 0.05 mumol/L and also a 5-HT4 antagonist at 1 mumol/L or more; and 0.01 mumol/L GR 113808, a new selective 5-HT4 antagonist. A chloride-free solution or furosemide (100 mumol/L) was used to show the relationship of a 5-HT-induced change in Isc to chloride secretion. RESULTS. Data were analyzed by ANOVA; $p < 0.05$ was significant. The chloride-free solution and furosemide significantly ($p < 0.05$) depressed the maximum change in Isc. Significant shifts occurred in the median effective concentration (1.5 +/- 0.2 mumol/L) for 5-HT in the presence of 1 mumol/L ICS 205-930 (3 +/- 0.2) and 0.03 mumol/L GR 113808 (2.4 +/- 0.2), but not in the presence of 5-HTP-DP (1.2 +/- 0.4), methysergide (1.8 +/- 0.3), ketanserin (2.4 +/- 0.6), ondansetron (1.6 +/- 0.1), 0.05 micron ICS 205-930 (1.3 +/- 0.1), or tetrodotoxin (1.4 +/- 0.4). CONCLUSIONS. In the human jejunum in vitro, a 5-HT-induced change in Isc is mediated through a tetrodotoxin-insensitive pathway by the 5-HT4 receptor. Antagonists to this receptor may be useful in the treatment of diarrhea in carcinoid syndrome.

L4 ANSWER 44 OF 52 MEDLINE on STN
ACCESSION NUMBER: 1990298258 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2141798
TITLE: Effects of 5-HT3 receptor antagonists on 5-HT and nicotinic depolarizations in guinea-pig submucosal neurones.
AUTHOR: Vanner S; Surprenant A
CORPORATE SOURCE: Vollum Institute, Oregon Health Sciences University, Portland 97201.
CONTRACT NUMBER: NS 25996 (United States NINDS NIH HHS)
SOURCE: British journal of pharmacology, (1990 Apr) Vol. 99, No. 4, pp. 840-4.
Journal code: 7502536. ISSN: 0007-1188.
Report No.: NLM-PMC1917554.
PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: (IN VITRO)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199008
ENTRY DATE: Entered STN: 7 Sep 1990
Last Updated on STN: 7 Sep 1990
Entered Medline: 8 Aug 1990

AB 1. Intracellular recordings were made from neurones of the guinea-pig submucosal plexus. The effects of several 5-hydroxytryptamine3 (5-HT3) receptor antagonists on depolarizations produced by ionophoretic application of 5-HT and acetylcholine, as well as on fast excitatory postsynaptic potentials (fast e.p.s.ps) produced by nerve stimulation were examined. 2. ICS 205-930, GR 38032F, MDL 72222, cocaine and curare all inhibited the fast e.p.s.p. as well as the depolarizations in response to 5-HT and acetylcholine (ACh) ionophoresis in a dose-dependent fashion. 3. IC50 values for ICS 205-930, GR 38032F, MDL 72222, cocaine and curare in inhibiting the 5-HT mediated depolarizations were 12 nM, 100 nM, 3 microM, 3 microM and 20 microM, respectively. 4. IC50 values for ICS 205-930, GR 38032F, MDL 72222, cocaine and curare in inhibiting the nicotinic depolarizations were 4 microM, 12 microM, 11 microM, 6 microM and 17 microM, respectively. Similar IC50 values were obtained for inhibition of the fast e.p.s.ps by these antagonists. 5. The nicotinic receptor blocker, hexamethonium, inhibited the nicotinic depolarization and the fast e.p.s.p. with IC50 values of 10 microM. Hexamethonium (10 microM-5 mM) did not alter the depolarization induced by 5-HT. 6. These results demonstrate that the pharmacological profile of 5-HT3 receptors present on submucosal neurones is identical to that of 5-HT3 receptors on myenteric neurones and, thus, provide evidence that the enteric neuronal 5-HT3 receptor forms a receptor subtype distinct from that characterized in other parts of the autonomic nervous system.

L4 ANSWER 45 OF 52 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:79521 BIOSIS
DOCUMENT NUMBER: PREV200200079521
TITLE: Systemic pharmacomodulation of transient lower esophageal sphincter relaxations.
AUTHOR(S): Holloway, Richard H. [Reprint author]
CORPORATE SOURCE: Department of Gastroenterology, Hepatology, and General Medicine, Royal Adelaide Hospital, Department of Medicine, University of Adelaide, Adelaide, South Australia, Australia
SOURCE: American Journal of Medicine, (December 3, 2001) Vol. 111, No. Supplement 8A, pp. 178S-185S. print.
CODEN: AJMEAZ. ISSN: 0002-9343.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jan 2002
Last Updated on STN: 25 Feb 2002

AB Transient lower esophageal sphincter relaxations (TLESRs) are the major mechanism of reflux in patients with gastroesophageal reflux disease. They are therefore attractive targets for pharmacotherapy. During the past 5 years, there has been a burgeoning interest in the neural pathways that control these events and in the pharmacologic receptors involved in these pathways. Several agents have been shown to reduce the rate of TLESRs, including cholecystokinin-A antagonists, anticholinergic agents, nitric oxide synthase inhibitors, morphine, somatostatin, serotonin type 3-receptor antagonists, and gamma-aminobutyric acid-B (GABAB) agonists.

Their predominant site of action appears to be on either the afferent pathways and/or the central integrative mechanisms within the dorsal vagal complex in the brainstem. Most of the agents tested are unsuitable for clinical use either because of side effects or because of the lack of an orally effective formulation. The most promising agents identified to date are the GABAB agonists. Baclofen, the prototype GABAB agonist, inhibits the rate of TLESRs by more than 50%. Control of TLESRs is a major new approach to the treatment of reflux disease. It is likely to be applicable to the majority of patients, particularly those without macroscopic mucosal lesions or only mild erosive disease. Further development of more effective agents will depend both on a better understanding of the neural pathways and receptors involved in the control of TLESRs, as well as on investigation of other novel agents. At present, inhibition of TLESRs is at the threshold of transition from concept to practical use. Whether it makes the final leap into the mainstream of therapy will depend on the development of new, novel, and well-targeted pharmacologic agents.

L4 ANSWER 46 OF 52 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002421542 EMBASE
TITLE: Inhibitory interactions between 5-HT(3) and P2X channel in submucosal neurons.
AUTHOR: Barajas-Lpez, Carlos (correspondence); Montano, Luis M.; Espinosa-Luna, Rosa
CORPORATE SOURCE: Botterell Hall, Queen's Univ., Kingston, Ont. K7L 3N6, Canada. barajasc@meds.queensu.ca
SOURCE: American Journal of Physiology - Gastrointestinal and Liver Physiology, (1 Dec 2002) Vol. 283, No. 6 46-6, pp. G1238-G1248.
Refs: 39
ISSN: 0193-1857 CODEN: APGPDF
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 12 Dec 2002
Last Updated on STN: 12 Dec 2002

AB Inhibitory interactions between 5-HT subtype 3 (5-HT(3)) and P2X receptors were characterized using whole cell recording techniques. Currents induced by 5-HT (I(5-HT)) and ATP (I(ATP)) were blocked by tropisetron (or ondansetron) and pyridoxalphosphate-6-azophenyl-2',4'-disulfonic acid, respectively. Currents induced by 5-HT + ATP (I(5-HT+ATP)) were only as large as the current induced by the most effective transmitter, revealing current occlusion. Occlusion was observed at membrane potentials of -60 and 0 mV (for inward currents), but it was not present at +40 mV (for outward currents). Kinetic and pharmacological properties of I(5-HT+ATP) indicate that they are carried through 5-HT(3) and P2X channels. Current occlusion occurred as fast as activation of I(5-HT) and I(ATP), was still present in the absence of Ca(2+) or Mg(2+), after adding staurosporine, genistein, K-252a, or N-ethylmaleimide to the pipette solution, after substituting ATP with α,β-methylene ATP or GTP with GTP-γ-S in the pipette, and was observed at 35°C, 23°C, and 8°C. These results are in agreement with a model that considers that 5-HT(3) and P2X channels are in functional clusters and that these channels might directly inhibit each other.

L4 ANSWER 47 OF 52 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2002302681 EMBASE
TITLE: Cosensitivity of vagal mucosal afferents to histamine and 5-HT in the rat jejunum.
AUTHOR: Kreis, M.E.; Jiang, W.; Kirkup, A.J.; Grundy, D. (correspondence)
CORPORATE SOURCE: Univ. of Sheffield, Dept. of Biomedical Science, Western Bank, Sheffield S10 2TN, United Kingdom.
SOURCE: American Journal of Physiology - Gastrointestinal and Liver Physiology, (Sep 2002) Vol. 283, No. 3 46-3, pp. G612-G617. Refs: 22
ISSN: 0193-1857 CODEN: APGPDF
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 002 Physiology
029 Clinical and Experimental Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 13 Sep 2002
Last Updated on STN: 13 Sep 2002

AB A complex sensitivity of afferent nerves in the mesentery of the rat jejunum to systemic administration of histamine has recently been demonstrated. In the present study, we aimed to characterize subpopulations of mesenteric afferents that mediate this afferent nerve response. Multiunit afferent discharge was recorded from mesenteric nerves supplying the proximal jejunum in anesthetized rats. The majority of mesenteric bundles (84%) exhibited biphasic responses to histamine (8 μmol/kg), and these bundles also responded to 2-methyl-5-HT (2m5HT). In contrast, monophasic responses lacked a short-latency component, and these bundles failed to respond to 2m5HT. Single-unit analysis revealed a population of afferents that possessed cosensitivity for 2m5HT and histamine. This population of afferents was absent in chronically vagotomized animals, whereas mucosal anesthesia with luminal lidocaine reversibly converted the biphasic profile to a monophasic one. Ondansetron (500 μg/kg) blocked the response to 2m5HT with no effect on the profile of the histamine response, whereas pyrilamine (5 mg/kg) blocked the histamine response without affecting the response to 2m5HT. We conclude that histamine-sensitive afferents exist in the rat proximal jejunum that also respond to 5-HT via the 5-HT(3) receptor. These fibers appear to be vagal afferents originating in the intestinal mucosa and may be involved in the organization of mast cell-mediated responses.

L4 ANSWER 48 OF 52 EMBASE
ACCESSION NUMBER: 2002013874 EMBASE
TITLE: Systemic pharmacomodulation of transient lower esophageal sphincter relaxations.
AUTHOR: Holloway, Richard H., Dr. (correspondence)
CORPORATE SOURCE: Department of Gastroenterology, Hepatology, and General Medicine, Royal Adelaide Hospital, University of Adelaide, Adelaide, SA, Australia.
AUTHOR: Holloway, Richard H., Dr. (correspondence)
CORPORATE SOURCE: Department of Gastroenterology, Royal Adelaide Hospital, University of Adelaide, Adelaide, Australia.
SOURCE: American Journal of Medicine, (3 Dec 2001) Vol. 111, No. 8 SUPPL. 1, pp. 178S-185S. Refs: 61
ISSN: 0002-9343 CODEN: AJMEAZ
PUBLISHER IDENT.: S 0002-9343(01)00853-1
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)

FILE SEGMENT: 037 Drug Literature Index
038 Adverse Reactions Titles
048 Gastroenterology
006 Internal Medicine
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Jan 2002
Last Updated on STN: 17 Jan 2002

AB Transient lower esophageal sphincter relaxations (TLESRs) are the major mechanism of reflux in patients with gastroesophageal reflux disease. They are therefore attractive targets for pharmacotherapy. During the past 5 years, there has been a burgeoning interest in the neural pathways that control these events and in the pharmacologic receptors involved in these pathways. Several agents have been shown to reduce the rate of TLESRs, including cholecystokinin-A antagonists, anticholinergic agents, nitric oxide synthase inhibitors, morphine, somatostatin, serotonin type 3-receptor antagonists, and γ-aminobutyric acid-B (GABA(B)) agonists. Their predominant site of action appears to be on either the afferent pathways and/or the central integrative mechanisms within the dorsal vagal complex in the brainstem. Most of the agents tested are unsuitable for clinical use either because of side effects or because of the lack of an orally effective formulation. The most promising agents identified to date are the GABA(B) agonists. Baclofen, the prototype GABA(B) agonist, inhibits the rate of TLESRs by more than 50%. Control of TLESRs is a major new approach to the treatment of reflux disease. It is likely to be applicable to the majority of patients, particularly those without macroscopic mucosal lesions or only mild erosive disease. Further development of more effective agents will depend both on a better understanding of the neural pathways and receptors involved in the control of TLESRs, as well as on investigation of other novel agents. At present, inhibition of TLESRs is at the threshold of transition from concept to practical use. Whether it makes the final leap into the mainstream of therapy will depend on the development of new, novel, and well-targeted pharmacologic agents. .COPYRGT. 2001 by Excerpta Medica, Inc.

L4 ANSWER 49 OF 52 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2001360241 EMBASE
TITLE: Intestinal serotonin acts as paracrine substance to mediate pancreatic secretion stimulated by luminal factors.
AUTHOR: Li, Y. (correspondence); Wu, X.Y.; Zhu, J.X.; Owyang, C.
CORPORATE SOURCE: Division of Gastroenterology, Univ. of Michigan, 6510 Med. Sciences Research Bldg. I, 1150 West Medical Center Dr., Ann Arbor, MI 48109-0682, United States. yli@umich.edu
SOURCE: American Journal of Physiology - Gastrointestinal and Liver Physiology, (2001) Vol. 281, No. 4 44-4, pp. G916-G923. Refs: 49
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COUNTRY: United States
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FILE SEGMENT: 002 Physiology
048 Gastroenterology
LANGUAGE: English
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AB We recently demonstrated that luminal factors such as osmolality, disaccharides, and mechanical stimulation evoke pancreatic secretion by activating 5-hydroxytryptamine subtype 3 (serotonin-3, 5-HT(3)) receptors on mucosal vagal afferent fibers in the intestine. We

hypothesized that 5-HT released by luminal stimuli acts as a paracrine substance, activating the mucosal vagal afferent fibers to stimulate pancreatic secretion. In the in vivo rat model, luminal perfusion of maltose or hypertonic NaCl increased 5-HT level threefold in intestinal effluent perfusates. Similar levels were observed after intraluminal 10(-5) M 5-HT perfusion. These treatments did not affect 5-HT blood levels. In a separate study, intraduodenal, but not intraileal, 5-HT application induced a dose-dependent increase in pancreatic protein secretion, which was not blocked by the CCK-A antagonist CR-1409. Acute vagotomy, methscopolamine, or perivagal or intestinal mucosal application of capsaicin abolished 5-HT-induced pancreatic secretion. In conscious rats, luminal 10(-5) M 5-HT administration produced a 90% increase in pancreatic protein output, which was markedly inhibited by the 5-HT(3) antagonist ondansetron . In conclusion, luminal stimuli induce 5-HT release, which in turn activates 5-HT(3) receptors on mucosal vagal afferent terminals. In this manner, 5-HT acts as a paracrine substance to stimulate pancreatic secretion via a vagal cholinergic pathway.

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ACCESSION NUMBER: 1999136280 EMBASE
TITLE: Aspects on reducing gastrointestinal adverse effects associated with radiotherapy.
AUTHOR: Henriksson, Roger, Dr. (correspondence); Bergstrom, Per; Franzen, Lars
CORPORATE SOURCE: Departments of Oncology, Umea University Hospital, Sodersjukhuset (South Hospital), Stockholm, Sweden.
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AUTHOR: Henriksson, Roger, Dr. (correspondence)
CORPORATE SOURCE: Department of Oncology, University Hospital, S-901 85 Umea, Sweden.
SOURCE: Acta Oncologica, (1999) Vol. 38, No. 2, pp. 159-164. Refs: 37
ISSN: 0284-186X CODEN: ACTOEL
COUNTRY: Norway
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
016 Cancer
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
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AB Patients receiving cancer therapy are afflicted with a diversity of side effects. Radiotherapy for cancer affecting the head and neck, oesophagus and pelvis is associated with a marked toxicity, specifically encountered as mucosal toxicity. Pain and diarrhoea as well as nausea and vomiting are the most common symptoms, with subsequent problems such as malnutrition and decreased quality of life. These side effects need to be reduced if we are to optimize radiotherapy and to cure patients. Because there is no straightforward way of obviating these side effects, every effort to prevent aggravation and to induce healing of mucosal changes is of prime importance. Numerous agents including antimicrobials, local and systemic analgesics, anti-inflammatory drugs, anti-diarrhoeal drugs, and mucosal protectors alone or in combination with dietetic care have been used and/or are under evaluation in order to palliate the symptoms and increase the quality of life for the patients subjected to radiotherapy. In this article we summarize some aspects

within the field that were discussed at the Annual Meeting of the Swedish Society for Oncology in Gavle, 1997.

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ACCESSION NUMBER: 1998343817 EMBASE
TITLE: Oral transmucosal fentanyl [2].
AUTHOR: Prosser, D. (correspondence); Allman, M.; Grassby, P.
CORPORATE SOURCE: Royal Gwent Hospital, Newport, Gwent, United Kingdom.
SOURCE: Anaesthesia, (1998) Vol. 53, No. 10, pp. 1030.
Refs: 3
ISSN: 0003-2409 CODEN: ANASAB
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Letter
FILE SEGMENT: 024 Anesthesiology
037 Drug Literature Index
038 Adverse Reactions Titles
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007 Pediatrics and Pediatric Surgery
LANGUAGE: English
ENTRY DATE: Entered STN: 28 Oct 1998
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ACCESSION NUMBER: 1995318839 EMBASE
TITLE: Comparative adverse effect profiles of platinum drugs.
AUTHOR: McKeage, M.J., Dr. (correspondence)
CORPORATE SOURCE: Oncology Research Centre, Prince of Wales Hospital, University of New South Wales, High St, Sydney, NSW 2031, Australia.
SOURCE: Drug Safety, (1995) Vol. 13, No. 4, pp. 228-244.
ISSN: 0114-5916 CODEN: DRSAEA
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
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037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
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AB Since the discovery of the biologically active platinum complexes 30 years ago, 2 agents have become widely established in clinical oncology practice. Both cisplatin and carboplatin are platinum(II) complexes with 2 ammonia groups in the cis- position. However, they differ in their solubility, chemical reactivity, dichloride or alicyclic oxygenated leaving groups, pharmacokinetics and toxicology. Cisplatin causes severe renal tubular damage and reduces glomerular filtration, and requires concurrent saline hydration and mannitol diuresis to eliminate potentially lethal and unacceptable damage to the kidneys. Carboplatin, at conventional doses, causes no decrease in glomerular filtration and only minor transient elevations in urinary enzymes. Cisplatin is the most emetic cancer drug in common use, while nausea and vomiting associated with carboplatin are moderately severe. Serotonin release from enterochromaffin gut mucosal cells and stimulation of serotonin 5-HT(3)-receptors mediates acute emesis. Selective inhibitors of the 5-HT(3)-receptor protect against cisplatin- and carboplatin-induced nausea and vomiting. Peripheral neurotoxicity is the most dose-limiting problem associated with cisplatin. Loss of vibration sense, paraesthesia and sensory ataxia comes on after several treatment cycles. Carboplatin,

however, is relatively free from peripheral neurotoxicity. Audiometry shows cisplatin-induced ototoxicity in 75 to 100% of patients, which may be associated with tinnitus and hearing loss. Ototoxicity is rare with conventional dose carboplatin therapy. Monitoring hearing with audiograms may identify early signs before significant impairment occurs. Cisplatin causes mild haematological toxicity to all 3 blood lineages. Haematological toxicity is dose-limiting for carboplatin, with thrombocytopenia being a greater problem than leucopenia. Although carboplatin is not toxic to the kidney, renal function markedly affects the severity of carboplatin-induced thrombocytopenia. The major clearance mechanism of cisplatin is irreversible binding in plasma and tissues, while carboplatin is cleared by glomerular filtration. Metabolism of cisplatin to aqua, amino acid and protein species is extensive, whereas carboplatin exists mainly as the free unchanged form. Strong relationships between carboplatin renal clearance, glomerular filtration rate, area under the plasma concentration-time curve (AUC) of filterable platinum and severity of thrombocytopenia have prompted dose adjustment according to renal function. New analogues such as JM216 offer the potential advantages of oral administration and few nonhaematological toxicities. Analogues based on the diaminocyclohexane ligand have encountered problematic neurotoxicity.

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